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E XVI, NUMBER 754 JANUARY 1940 TABLE OF CONTENTS, PAGE II

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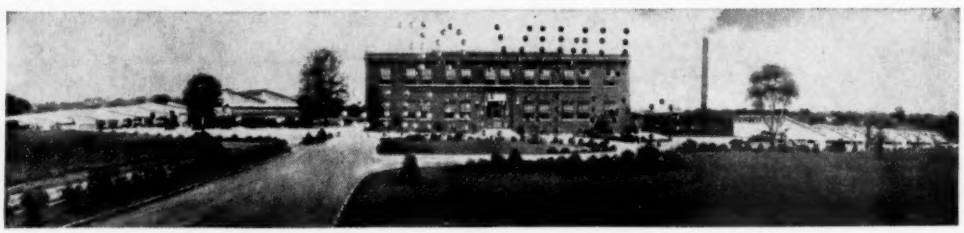

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Poultry Remedies Under the New Food, Drug, and Cosmetic Act*

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Washington, D. C.

THERE ARE more poultry remedies on the market than all other veterinary remedies combined, in spite of the fact that the therapeutic value of internal medication for poultry is extremely limited.

Interstate shipments of medicinal preparations for poultry have been subject to the requirements of the Food and Drugs Act of 1906, and now are subject to the provisions of the new Food, Drug, and Cosmetic Act of June 25, 1938. Insecticidal preparations for poultry, including disinfectants, are subject to the requirements of the Federal Insecticide Act of 1910. However, these acts have no jurisdiction over products manufactured and sold locally, and the Administration has no authority under these acts to regulate collateral advertising matter distributed separately from the product. The recently enacted Lee-Wheeler Act, which deals with the advertising of foods, drugs, and cosmetics for man and animals, is being enforced by the Federal Trade Commission. The advertising and shipping of poultry remedies through the mails are subject to the postal laws enforced by the postoffice department.

*Presented before the Section on Poultry, 76th annual meeting of the A.V.M.A., Memphis, Tenn., August 28 to September 1, 1939.

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TERM "DEVICE" IS DEFINED IN 1938 STATUTE

There are several new provisions of the Food, Drug, and Cosmetic Act, which is being enforced by the Food and Drug Administration, that have an important bearing on interstate shipments of poultry remedies. The term "device" under the new act is defined to mean, "instruments, apparatus, and contrivances, including their components, parts, and accessories, intended for use in the diagnosis, cure, mitigation, treatment, or prevention of disease in man or other animals; or to affect the structure or any function of the body of man or other animals." Therefore, articles such as surgical instruments and anti-picking devices for poultry come within the definition of the term "device." Products represented for increasing weight, egg production and fertility also come within this definition, in connection with their effect on the structure and function of the body.

NEW ACT RIGIDLY GOVERNS LABELING OF INTERSTATE-COMMERCE PRODUCTS

Any manufacturer who intends to place a new drug in the channels of interstate commerce is required under the act to file an application with the Secretary of Agri-

culture, giving a full report of investigations which have been made to show whether or not such drug is safe for use. Under the terms of this new act, interstate shipments of drugs which are dangerous to the health of poultry when used in the dosage or with the frequency or duration prescribed, recommended, or suggested in the labeling thereof are prohibited. The act, in general, requires the labeling of drugs and devices to bear warnings against probable misuse which may be dangerous to health; it also requires special precautionary labeling for drugs that are liable to deteriorate. The act also prohibits interstate traffic in drugs that have been prepared or handled under insanitary conditions whereby they may have been contaminated with filth or that may have rendered the contents injurious to health. Drugs and devices when shipped in interstate commerce must bear the name and place of business of the manufacturer, packer, or distributor. The labeling of a drug must bear an accurate statement of the quantity of the contents, and the common or usual name of each active ingredient and, in addition, the quantity or proportion of certain specified substances. While the effective date of this section of the act, which requires the declaration of the common or usual name of each active ingredient, is postponed until January 1, 1940, when it does become effective, it will be a distinct advantage to veterinarians and the public to know of what active ingredients the product is composed. Veterinarians, particularly, will be in a position to judge the therapeutic value of such articles in most instances without an actual analysis.

The act limits seizure for misbranding to a single interstate shipment of the product, unless the misbranding has been the subject of a prior court decision in favor of the government, or unless the misbranded article is dangerous to health, or its labeling is fraudulent or would be in a material respect misleading, to the jury or damage of the purchaser or consumer. However, the new act for the first time authorizes the federal courts to restrain violations by injunction. One of the most important factors of the new act from the standpoint of therapeutic claims is that a drug or device is misbranded if its labeling is false or misleading in any particular. It will, therefore, be no longer required by the government to prove that false claims of curative effect on the labels of drugs were made with willful intent to deceive, as was required under the Food and Drugs Act of 1906. No attempt has been made here to cover all of the requirements of the new act as they apply to poultry remedies. More specific information can be obtained by consulting a copy of the act and the regulations promulgated for its enforcement.

An efficient poultryman naturally wants his poultry to be healthy. This desire, when not combined with a proper knowledge of what constitutes a reliable treatment or cure for certain poultry diseases, has led poultrymen to spend much hard-earned money for remedies of no value whatever in the treatment of poultry diseases. While brief mention will be made of some of the activities of the Food and Drug Administration in the past in the enforcement of the Food and Drugs Act of 1906 against misbranded poultry remedies, the writer does not want to be understood as condemning all poultry remedies or all medicine manufacturers. The Food and Drugs Act of 1906, as well as the new act, fully recognizes the right of manufacturers of honestly labeled preparations to do business. There are many manufacturers who are earnestly seeking literally to comply with the law.

MISREPRESENTED POULTRY REMEDIES OUTLAWED FROM INTERSTATE CHANNELS

The Food and Drug Administration in coöperation with the U. S. bureau of animal industry has tested many poultry-worm remedies on the market. None of the products tested up to the present time has shown any value in removing any species of tapeworms, including the heads, from poultry. The Administration has, therefore, been active in the removal of these types of preparations from interstate channels as rapidly as possible. As a re-

sult of our activity in this connection there has been a tendency on the part of some manufacturers to represent their articles as being effective for removing segments of tapeworms. According to veterinary parasitologists, the only scientific evidence available on the injurious effect of tapeworms is through means of the head, which embeds itself in the mucous membrane of the intestine, thereby interfering with the physiological function of the intestinal tract. Mature segments of tapeworms are naturally passed to propagate the species, and there is no scientific evidence that the removal of segments of tapeworms by the use of a drastic cathartic is of any value to the bird. Under the circumstances, the Administration regards the use of statements such as "to remove the segments of tapeworms" or "desegmenting tapeworms" as being misleading and, therefore, objectionable from the standpoint of the law. In our tests we have found that many of the products on the market containing the alkaloid nicotine or nicotine sulfate are not sufficiently effective to justify the claims for removing large roundworms from poultry. From our knowledge of conducting these tests, it is apparent that the effective dose of nicotine alkaloid for grown chickens is approximately .0486 Gm. (0.75 gr.), or .0972 Gm. (1.5 gr.) of nicotine sulfate, to remove large roundworms. Under no condition have we found any single dose treatment by way of the mouth to be effective for the removal of cecal worms from poultry. The incorporation of known effective anthelmintics in the feed has not proved entirely satisfactory in our experience. It appears that birds which are heavily infested with large roundworms consume less of the medicated mash than those which have little or no infestation.

FOOD AND DRUG ADMINISTRATION SHOWS COCCIDIOSIS REMEDIES TO BE VALUELESS

Many coccidiosis remedies have appeared on the market from time to time. Our tests of these remedies up to the present time, including the widely advertised product known as Red-Hed Coxol, manufactured by Production Laboratories, Seattle, Wash., have shown that they are of no value whatever. These remedies are popular undoubtedly because of the fact that spontaneous recoveries may occur in a flock of chicks affected with coccidiosis where proper attention has been given to preventing the chicks from reinfecting themselves from coccidia passed in the droppings, or to the resistance or immunity established in the chicks from a light infection. Recently, we tested a preparation named Coc-Ci-Tox, which was represented as a preventive or control of coccidiosis by treating the litter. This product, which was found upon analysis to consist of sodium nitrate with blue coloring, proved to be of no value when used as directed. The reported experimental work with the feeding of sulfur has shown that it has no value in the treatment of chicks infected with coccidiosis and that the amount of sulfur which is necessary for the prevention of coccidiosis infection is too injurious to warrant its use. Unfortunately, the publication of this information has resulted in the placing of sulfur products on the market under many unwarranted and pseudoscientific claims. The Administration at the present time, in the enforcement of the Food, Drug, and Cosmetic Act, is investigating one of these products. We have already received information that at least three state institutions have tested the product under controlled conditions and found it to be of no value in the control of coccidiosis.

TERM "CONDITIONER" IS BANNED FROM FOOD AND DRUG LABELS

Just how the term "conditioner" for drugs and food products for animals originated, the writer does not know. It does not appear to be used to any great extent for drugs and foods for human use. Its extensive use, and abuses, for veterinary remedies are well known. Since there is no known drug or combination of drugs or foods that justifies its inclusion, the Food and Drug Administration, in the enforcement of the Food, Drug, and Cosmetic Act, can not sanction its appearance

in connection with the labeling of drugs and foods.

MISCELLANEOUS COMMENTS

Some time ago, there was an article published in a popular poultry publication dealing with the treatment of respiratory diseases of poultry with hypochlorite powder, based on some experimental work conducted by a veterinarian at a state experiment station in the East. According to the paper, the effectiveness of the treatment is the result of the inhalation of chlorine gas liberated from the powder. At the World's Poultry Congress there was presented a foreign paper, read before the disease section, dealing with the treatment of respiratory diseases with chlorine gas. This writer is reliably informed by medical authorities that the chlorine-gas treatment formerly advocated by some army physicians has been abandoned as being of little or no value in the treatment of colds or any other respiratory disease of man. It has been the writer's personal experience that the irritating properties from the inhalation of chlorine gas will do more harm than good to the mucous membrane of the upper respiratory tract. From a consideration of the pathology and cause of the specific disease conditions involving the respiratory tract of poultry, it does not seem possible that such treatments could be efficacious in controlling any of the specific infectious diseases involving the respiratory tract of poultry.

For some reason, the various forms of phenolsulfonates have become popular for the treatment of disease conditions of poultry and other animals involving the intestinal tract. The theory appears to be based on the assumption that such products are broken down in the intestinal tract with the liberation of phenol and, for this reason, such products are thought to have antiseptic action in the intestinal tract. However, it is well known to veterinary and medical authorities that there is no known drug or mixture of drugs which can be depended upon to inhibit the bacterial flora of the intestinal tract. Bacteriological tests conducted by the Food and Drug Administration with sodium and calcium phenolsulfonates have shown definitely that they have no germicidal or antiseptic activity, and while zinc phenolsulfonate has some inhibitory action in concentrated form, in the dilutions recommended for poultry it was not found to have any germicidal or inhibitory effect.

Information appearing in textbooks and elsewhere dealing with certain drugs as respiratory and intestinal antiseptics is obsolete in the light of our present scientific knowledge and should be deleted from such publications. In many instances it seems that sufficient work of a scientific character has not been done to justify some of the definite claims made for drug products in textbooks and other scientific periodicals. Manufacturers frequently make use of such literature, as well as of testimonials and other printed information from agricultural extension agents, veterinarians, and others, to justify the claims made for their products. It has been our experience that a great deal of this information on the therapeutic value of drugs is often based upon information obtained from clinical results interpreted by those who do not have a thorough knowledge of the disease with which they are dealing. Until such time as drugs have been proved definitely, from the standpoint both of the scientific worker and the clinician, to be effective in the treatment of diseases, the propriety of such publications seems doubtful.

If veterinarians desire to coöperate with the Food and Drug Administration in removing misbranded products from interstate traffic, they should exercise due care before recommending in scientific articles or other publications any drug or combination of drugs for the treatment of diseases of poultry or other animals.

No scientific discovery has ever aroused more general interest than Pasteur's demonstrations on the cause of fermentation (1857).

Hepatic Cirrhosis of Horses, Swine and Cattle Due to the Ingestion of Seeds of the Tarweed, Amsinckia Intermedia*

By ERNEST C. McCULLOCH, D.V.M., M.A., Ph.D.
Pullman, Wash.

ENZOÖTIC hepatic cirrhosis of horses in the wheat-producing regions of the Pacific Northwest has been described by Kalkus, Trippeer, and Fuller,[1] who also stated that "a similar condition which may have a like origin is occasionally encountered in cattle and is seen not uncommonly in hogs." "Autopsy reveals a cirrhotic liver," the authors observed, "but clinical delirium is not manifested in hogs."

The condition in horses is locally called "walking disease" because of the tendency of many of the affected animals to wander aimlessly until death. In swine the names most frequently applied are "hard liver disease" or "Walla Walla hard liver disease" because of the dense fibrotic condition of the affected livers and because many cases have been observed in Walla Walla county (Wash.). In certain sections of Idaho and Oregon the condition is more generally known as protein poisoning or winter-wheat poisoning because its occurrence has been associated with the feeding of certain diets, especially those high in winter wheat.

SYMPTOMATOLOGY

The symptoms of walking disease in horses were ably described by Kalkus, Trippeer, and Fuller[1] in 1925, who wrote:

> Probably the earliest recognizable symptom is a slight yellowness of the mucosae of the eyes, nose and mouth and a refusal to eat grain. In working horses, the coat may appear unthrifty, does not shed readily and there is a history of sluggishness. In some cases these premonitory symptoms do not develop and the first symptom may be a furious delirium, followed by death in a few hours or days. Such cases would be clinically classed as acute, but autopsy reveals that they are of long standing in so far as lesions are concerned.
>
> In the chronic cases the condition seems to come on more gradually. The visible mucous membranes may become a distinct yellow, although they may at times show congestion. In a case of long standing the membranes may clear up entirely for a time. Small, irregularly shaped ulcers usually develop on the mucous membrane of the lips and gums and the inside of the mouth may have an offensive, sour odor.
>
> As the disease progresses, a gradual emaciation develops. The horse shows an unsteady gait and appears sluggish, sleepy, and stupid. If it is tied, it will swing in the halter or lean against the manger. If loose, it leans against any convenient object, such as a fence, building or post. If there is no object near, the animal shifts its position frequently from one foot to the other and occasionally takes a step or two in order not to lose its balance. The animal is observed to gape and yawn frequently; it continues to eat hay, but the appetite seems impaired as it does not show the same desire for food as a normal horse, and refuses to eat grain. . . . As the disease advances many affected horses become restless and desire to travel; they walk straight ahead, colliding with objects and walking through fences. When in the open country, they walk aimlessly for miles.

In swine the nervous symptoms are less pronounced, although unsteadiness of gait and a tendency to walk aimlessly have been observed in some chronic cases. More frequently, the first symptom observed is a failure to make normal gains. After fairly normal growth to 14, 18, or even 45 kg. (30, 40 or 100 lbs.), the gains cease, the body becomes narrow, and the head appears elongated. The hair is rough and the pig presents an appearance typical of acute parasitism, although autopsies have revealed very few intestinal parasites. Later, the animal becomes icteric and sometimes cachectic. However, swine have been known to survive for over a year and, rarely, for two years after the first definite symptoms were shown.

Hard liver disease in cattle occurs much less frequently in this area and usually presents no symptoms other than lack of condition, a rough coat and some degree

*Published as scientific paper No. 423, College of Agriculture and Agricultural Experiment Station, State College of Washington.

Fig. 1. Pigs 1 and 3, March 30, 1939, after 105 days of experimental feeding. The white pig received A. intermedia; the black pig served as a control.

of icterus. Usually, the condition is recognized first in the abattoir.

OCCURRENCE

The condition has remained enzoötic in certain well-defined areas of eastern Washington and Oregon and western Idaho for many years, although both horses and swine have moved freely into surrounding counties. Heavy losses from walking disease are said to have occurred over 40 years ago. Few swine were raised in this region at that time and little attention was paid to swine diseases. Because of the substitution of tractors for horses in recent years, walking disease is now of less economic significance, while the importance of hard liver disease in swine and cattle has increased with the advent of diversified agricultural programs on grain ranches.

No spontaneous case of either disease has been reported in horses or swine reared and maintained in the vicinity of Pullman, Wash., which is located in the Palouse region and is approximately 40 miles from the nearest area where these conditions develop. Swine raised to weights of 18 to 34 kg. (40 to 75 lbs.) in the hard-liver-disease regions and apparently in good condition when brought to the Palouse region for feeding have subsequently developed hard liver disease but did not transmit it to locally reared pigs with which they were placed. Some of the horses, which were taken from the Palouse region to the walking-disease areas for work or for winter feeding, developed walking disease either before or after their return to the Palouse region but did not transmit the condition to local horses.

In the walking-disease regions, horses maintained in stables and receiving the

Fig. 2. Pig 2, May 6, 1939, after 142 days on a ration containing A. intermedia seed. Such pigs may survive for months, growing slightly longer and higher and slowly losing weight.

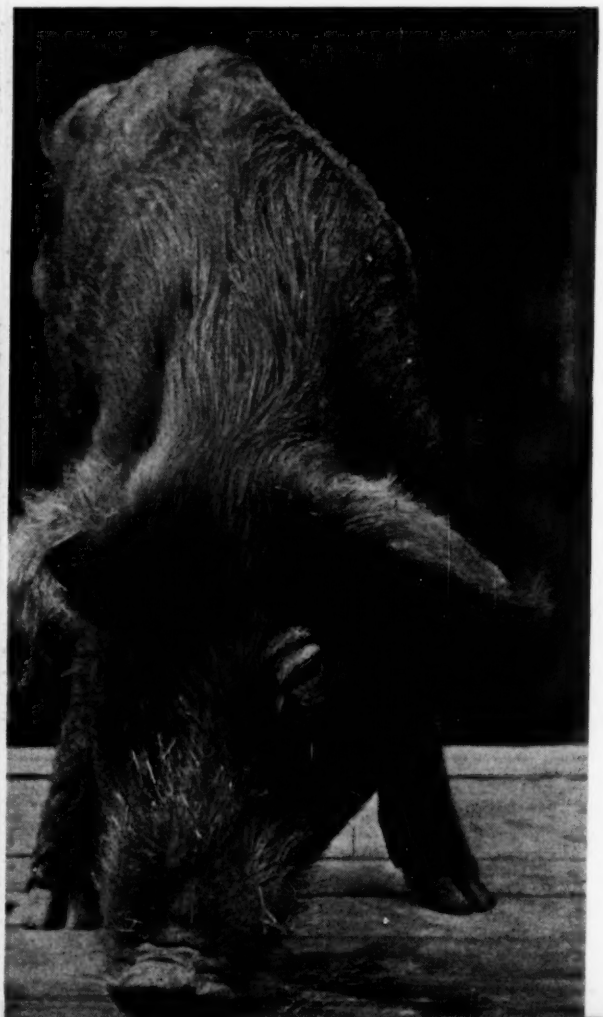

Fig. 3. Pig 3 (same as shown in fig. 1). This animal is typical of early fatal cases of amsinckiosis.

best of food and care rarely developed the condition. Also, the swine on certain farms have escaped hard liver disease over many years although heavy losses have occurred on adjoining farms. The disease has not been observed in horses maintained within the City of Walla Walla.

THEORIES OF THE ETIOLOGY OF WALKING DISEASE AND HARD LIVER DISEASE

The limitation of spontaneous cases almost entirely to regions sharply defined by the type of soil and amount of precipitation led to the theories that the disease is caused by some toxic element present in the soil or water, or that some insect vector peculiar to that environment spreads an etiological agent. The further limitation of the disease within these areas almost exclusively to farms growing and feeding the Triplet variety of winter wheat gave rise to the theory that perhaps, when fed in large amounts, the wheat itself is toxic or lacks some necessary element of nutrition. The prevalence of the condition in horses which were allowed to feed during the winter on stacks of wheat straw led to the belief that a mould growth on the wet straw was responsible, while the condition was sometimes diagnosed as botulism and treated with antitoxin. Parasitism, since many of the horses in this region are heavily infested with *Strongylus equinus* and the "small strongyles," especially members of the genus Cylicostomum (Trichonema), has been thought to have caused the condition. The administration of anthelmintics was sometimes followed by temporary improvement. Various plants, including *Amsinckia intermedia*, which is commonly known as fiddle neck, yellow burweed or yellow tarweed, also were suspected, although the irritant nature of the growing plants made it appear unlikely that animals would willingly eat them.

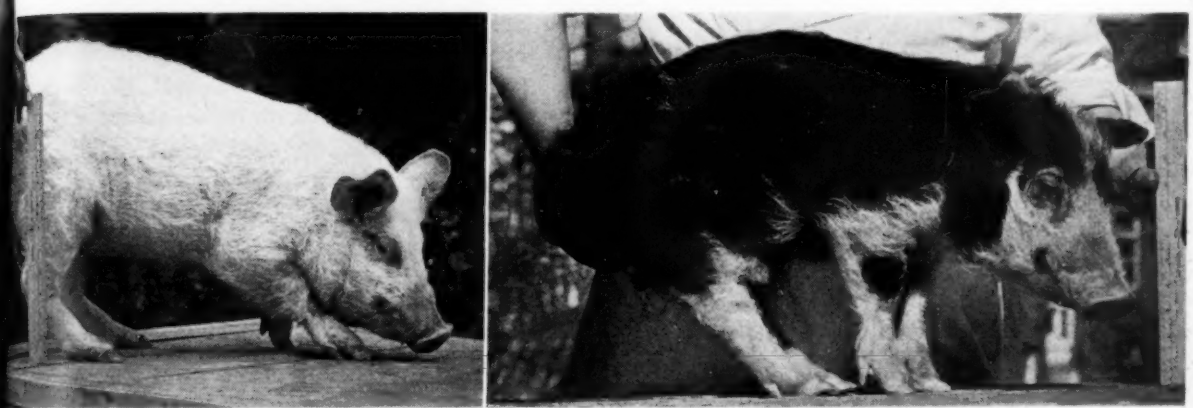

Fig. 4 (left). Pig. 4 after 162 days on a ration to which an aqueous infusion of A. intermedia seed had been added. Although growth was slow, due to the animal's refusal to eat an adequate amount of this treated ration, there was no evidence of amsinckiosis. Fig. 5 (right). Pig 5 on March 30, 1939, after 105 days on a ration containing A. intermedia seed and apples to supplement the diet. The animal had an extremely hard liver.

REVIEW OF LITERATURE

As early as 1906, Robertson[2] reported on hepatic cirrhosis of South African horses. He stated:

Fig. 6. Pig 6 after 142 days on a ration containing cooked A. intermedia seed. Although this pig is stunted, due to its refusal to consume a sufficient amount of the cooked seed, it did not have amsinckiosis.

Diseases in which cirrhosis of the liver is the most pronounced postmortem lesion are encountered over the greater part of the world and, in certain countries, have assumed the proportions of an epizoötic among stock. In the great majority of cases the cause has been traced to the ingestion of some irritant in the food or water.

In cases of chronic cirrhosis in the horse, or stomach staggers, to use its common name, the most common symptoms are: The horse is dull and drowsy-looking . . . and he may fall asleep with his head leaning on the manger and his mouth full of food . . . If his mouth is examined, by inserting the hand and smelling it when withdrawn, it will generally have an offensive, sour smell . . . If let out, he will wander on with an unsteady staggering gait, his head hanging down, and an unconscious glare about his eyes. By-and-by he becomes more excited and delirious and plunges about in a violent manner, with trembling of the muscles and apparent blindness . . . Or, he may become quite comatose, and die without a struggle.

The description of affected horses given by Kalkus, Trippeer, and Fuller[1] agrees quite well with these observations.

Much the same symptoms in horses were observed by Van Es, Cantwell, Martin, and Kramer[3] in Nebraska, who ably reviewed the literature on enzoötic hepatic cirrhosis up to 1929 and concluded:

> With the exception of the Schweinsberger disease of Germany and the walking disease of the Pacific Northwest, no enzoötic form of liver disease thus far described has occurred without its etiological relation with Senecio poisoning either having been definitely proved or seriously suspected.

These workers proved the walking disease *(necrobiosis et cirrhosis hepatis enzoötica)* of northwestern Nebraska to be due to the ingestion of *Senecio Riddellii.*

The "secondary" or "X" disease associated with equine encephalomyelitis and described by Madsen,[4] Marsh,[5] Shahan and coworkers[6] and others has many symptoms in common with enzoötic hepatic cirrhosis and these might cause confusion where the veterinarian observes the affected horses for the first time. The history, together with the extremely hard and cirrhotic liver of a horse poisoned with *A. intermedia* as compared with the friable liver found in "X" disease, should prove a sufficient basis for differentiation in those cases where an affected horse is available for autopsy.

RESULTS OF FIELD SURVEYS

Two careful surveys of the affected region in Walla Walla county, one made with William Harvey of the division of agronomy of the State College of Washington, and the other with W. T. Huffman and Ben S. Markham of the Bureau of Animal Industry, U. S. Department of Agriculture, failed to reveal the presence of members of the genus Senecio or other plants which are known to be extremely dangerous to animals.

The regions in which the condition occurs differ in the nature of the soil from the surrounding regions in that areas of fine, powdery dust, in which vegetation is lacking, occur on many hill sides. Samples of this soil, as well as of the tarweed, *A. intermedia,* which grows profusely in the regions, and of wheat from the affected region were forwarded to H. G. Byers, chief, Soil Chemistry and Physics, Bureau of Chemistry and Soils, U. S. Department of Agriculture, who examined them for selenium, which Beath and associates[7] and Moxom[8] found to be responsible for dullness, lack of vitality, emaciation, rough coat, and atrophy and cirrhosis of the liver. Four of the five samples contained 0.1 and one sample 0.2 parts of selenium per million.[9] This definitely eliminated selenium poisoning as the causative agent, since it is generally assumed that more than 10 parts of selenum per million must be present in a ration to produce recognizable pathological changes.

A. intermedia was observed to be much more prevalent in the regions from which walking disease and hard liver disease had been reported than in the surrounding areas in which the soil or precipitation were different. Fields of winter wheat, such as the Triplet variety (which had been associated with these conditions), were noticed to be most heavily infested

with *A. intermedia* because the weeds were not disturbed by spring plowing.

EXPERIMENTAL PROCEDURE

All attempts to produce walking disease in horses and hard liver disease in swine, cattle, sheep and rabbits were carried out at the College of Veterinary Medicine, State College of Washington. The feeding trials with rats were carried on in the nutrition laboratory of the division of home economics. All experimental animals were reared locally, either on the campus of the College or on nearby farms. Since the conditions under study never have occurred in the region of Pullman, their spontaneous occurrence during the course of experimentation is improbable. In most of the trials parallel controls were maintained, although expense prohibited this in the case of horses. However, the horses were maintained in the College veterinary hospital and no case of walking disease developed in the other horses kept there.

SWINE EXPERIMENTS

The basic ration fed the swine during these experiments consisted of:

Barley	80 kg.	(175 lbs.)
Peas	68 kg.	(150 lbs.)
Albit wheat	80 kg.	
Oats	136 kg.	(300 lbs.)
Alfalfa meal	68 kg.	
Tankage	22.7 kg.	(50 lbs.)
Steamed bone meal	4.5 kg.	(10 lbs.)
Salt	0.9 kg.	(2 lbs.)

This ration is commonly fed to the College swine herd. No case of hard liver disease has developed in this herd.

Six healthy, litter-mate, weanling pigs, 7 weeks old, weighing 8-9 kg. (18-20 lbs.), were purchased from a local farm, and four pigs, clinically typical of hard liver disease, were brought from a farm near Walla Walla. Two of the local weanling pigs were fed entirely on the basic ration but were exposed to any possible infectious agent of hard liver disease in the following ways: Two of the typical hard liver pigs were killed and the clinical diagnosis confirmed. Five cc. (0.17 oz.) of fresh blood from each of these was injected intraperitoneally into each of the two experimental pigs. Also, 5-gm. (0.17-oz.) portions of the liver and spleen of each of the hard liver pigs was macerated with sterile sand and physiological saline solution, the suspended material allowed to settle, and the supernatant fluid injected intraperitoneally into each of these experimental pigs. To provide additional exposure, the two remaining "hard liver" pigs were maintained in a box stall with the two experimental pigs. Hog lice (*=Hæmatopinus suis*) soon were transmitted from the affected pigs to the experimental pigs. During the early part of the experiment no attempt was made to control these parasites, since it was possible that they might transmit some infectious agent. The experimental pigs made practically normal gains and weighed 63 and 67 kg. (138 and 147 lbs.), respectively, when slaughtered after five months. Careful examination revealed no evidence of hepatic involvement or icterus.

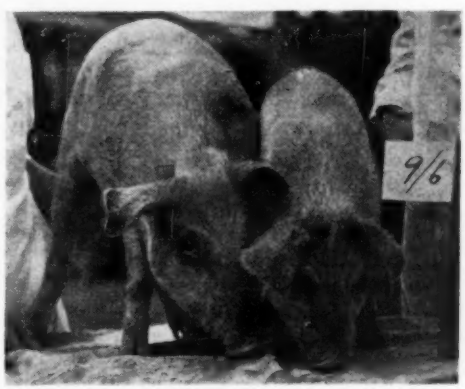

Fig. 7. Litter mates after 61 days of experimental feeding. The larger pig received cleaned Triplet wheat; the smaller pig was given the A. intermedia seed and broken grains of wheat removed from the cleaned wheat by screening. Both animals were killed, and the liver of the smaller pig showed damage typical of amsinckiosis. The larger pig was found to be normal upon autopsy.

The second two weanling pigs were inoculated with the blood and with material from the liver and spleen of the hard-liver-disease pigs, as were the first two, but were not maintained with hard-liver-disease pigs. They were fed two-thirds basic ration and one-third wheat screenings from the region of Walla Walla where hard

liver disease is prevalent. These screenings consisted of broken wheat, a few miscellaneous weed seeds and 10 to 25 per cent seed of *A. intermedia*. Both of these pigs soon developed symptoms typical of hard liver disease. One died after 112 days on this ration, and the other died after 156 days.

Autopsy revealed extremely cirrhotic livers and general icterus.

The exact amount of *A. intermedia* seed consumed was difficult to estimate because, while the pigs ate the seed fairly readily at first, they tended to avoid it after they first became ill. The pig that survived 156 days became quite adept in separating the ground basic ration and broken wheat in the screenings from the *A. intermedia* seed.

The third two pigs were isolated from the others, with no exposure to hard-liver-disease pigs. They were fed the same ration as the second pair—two-thirds basic ration and one-third wheat screenings. One of these pigs survived for 49 days, the other for 95 days. Both clinically and at autopsy the diagnosis of typical hard liver disease was confirmed. In the pig which died after 49 days, the liver was swollen, and the degree of icterus was even greater than that usually observed in these animals.

This series of experiments confirmed the suspicion that the etiological factor of hard liver disease is not transmissible but is contained in the wheat screenings.

The second series of feeding trials was started December 16, 1938. Six healthy pigs, weighing 8.2-10.5 kg. (18-23 lbs.) when put on experiment at the age of 8 weeks, were used.

Pig 1, weighing 8.2 kg., was used as a control and was fed the basic ration. At the end of three weeks it had lost 240 Gm. (8 oz.), but gained 3.8 kg. (8.5 lbs.) during the next twelve days and an additional 5.5 kg. (12 lbs.), during the following month. At the end of 105 days, this pig weighed 22 kg. (48 lbs.) and when butchered on May 17, weighed 36 kg. (79 lbs.). While growth in the experimental pens and on the basic ration alone was less than normal for a pig, its appearance was good, as is shown in figure 1, and upon autopsy no evidence of abnormal pathology could be found. The liver was normal and icterus was absent.

Pigs 2 and 3 each were fed equal parts of the basic ration and screenings. At the beginning of the experiment, pig 2 weighed 10.5 kg. (23 lbs.) and, three weeks later, 7.7 kg. (17 lbs.). This weight was maintained during the next twelve days. During the next month a gain of 3.2 kg. (7 lbs.) was made. At the end of 105 days it weighed 14 kg. (31 lbs.) and had assumed the appearance of a "hard liver" pig, as is shown in figure 2, taken after 142 days of experimental feeding. When killed at the end of 163 days, this animal weighed 17 kg. (37 lbs.). Pathological changes consisted of an extremely hard, cirrhotic liver and general icterus. This pig also became adept in avoiding the *A. intermedia* seed.

Pig 3 weighed 8.6 kg. (19 lbs.) when placed on experiment. It lost 2.3 kg. (5 lbs.) during the first three weeks and 480 Gm. (1 lb.) in the next twelve days. It gained 0.9 kg. (2 lbs.) during the next month and became very thin, as is shown in figures 1 and 3. Before it was killed in a moribund condition, April 3, it weighed 4.3 kg. (9.5 lbs.). Autopsy revealed an extremely hard, cirrhotic liver. Anemia was pronounced, and for that reason there was no evidence of icterus. Kalkus, Trippeer, and Fuller[1] had noted the disappearance of icterus in some horses shortly before death.

Pig 4 was fed the basic ration to which was added an aqueous infusion of ground screenings rich in *A. intermedia* seed obtained by mixing equal weights of the ground screenings and water, allowing it to stand over night, and pressing out the infusion. This was distasteful to the pig and only a minimum of the treated feed was consumed. At the beginning of the experiment, pig 4 weighed 8.2 kg. (18 lbs.) and lost 1.8 kg. (4 lbs.) during the first three weeks. It gained 0.9 kg. (2 lbs.) during the next twelve days and 4.1 kg. (9 lbs.) during the following month.

When killed on May 27, after 162 days of experimental feeding, this animal was stunted but otherwise apparently normal, as is illustrated in figure 4, and showed no gross evidence of hard liver disease in the liver. It weighed 17.3 kg. (38 lbs.).

Pig 5 was fed a ration consisting of equal parts of the basic ration and wheat screenings and, in addition, was given 100 to 300 Gm. (3.3 to 10 oz.) of fresh apples daily to ascertain whether the inclusion of succulence in the diet would prevent the development of hepatic cirrhosis. At the beginning, pig 5 weighed 8.6 kg. (19 lbs.) and lost 0.9 kg. (2 lbs.) during the first three weeks, 480 Gm. (1 lb.) during the next twelve days and gained 2.7 kg. (6 lbs.) in the next month.

This animal appeared to be in almost as good condition as the control during the first two months, then gradually assumed the appearance of a hard-liver-disease pig, as is shown in figure 5. It weighed 13.2 kg. (29 lbs.) when it was killed in a moribund condition, April 15, after 121 days of experimental feeding. Autopsy revealed a definitely cirrhotic liver and general icterus.

Pig 6 was fed equal parts of the basic ration and screenings which had been boiled for 15 minutes. The cooked screenings were objectionable to the pig and it frequently refused to consume them. At the beginning of the experiment this animal weighed 9 kg. (20 lbs.) and lost 2 kg. (4.4 lbs.) during the first three weeks. Its weight remained the same for the next twelve days and a gain of 1.6 kg. (3.5 lbs.) was made during the following month.

This animal assumed somewhat the appearance of a hard-liver-disease pig, as is shown in figure 6, but slowly was making gains and weighed 17.3 kg. (38 lbs.) when it was slaughtered after 162 days of feeding. While in poor flesh, the animal showed no gross evidence of hepatic cirrhosis or icterus.

These experiments confirmed the prior feeding trials and showed that the inclusion of wheat screenings, rich in the seeds of *A. intermedia* from the region where hard liver disease is enzoötic, would produce hepatic cirrhosis in swine. The inclusion of succulence, in the form of apples, failed to prevent the development of the condition. The amount of water extract of the screenings and of the cooked screenings fed failed to produce hepatic cirrhosis. Whether the toxic principle is not water soluble or whether an insufficient amount of the aqueous infusion was fed is not known. Neither is it known whether the boiling for 15 minutes destroyed the toxic principle.

An alcoholic extract obtained from approximately 480 Gm. (1 lb.) of *A. intermedia* seed, when administered orally to a 13.7-kg. (30-lb.) pig, caused death in less than twelve hours. The oily fraction of a similar extract failed to kill a 13.7-kg. pig while the nonoily fraction proved fatal to pigs.

TRIPLET WHEAT FOUND NONTOXIC

Because hard liver disease had long been associated with the feeding of Triplet and certain other varieties of winter wheat, a series of tests were planned to eliminate the possibility that the hepatic cirrhosis produced in the pigs in the previous experiments might have been due to the Triplet wheat present in the screenings.

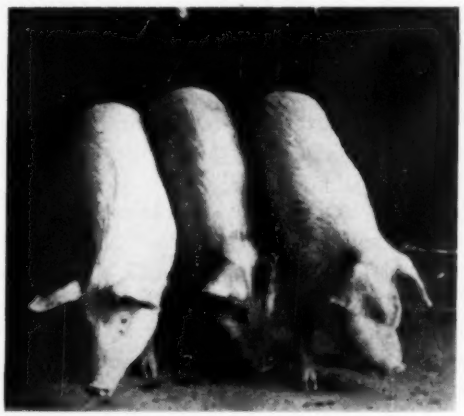

Fig. 8. Experimental animals after 110 days of feeding on cleaned Triplet wheat from Walla Walla. When slaughtered, all were normal. The postmortem findings proved that Triplet wheat from the "hard liver" region was not responsible for hepatic cirrhosis.

Triplet wheat from a farm on which

walking disease had occurred in the horses and hard liver disease in the swine was cleaned to remove the weed seeds and used in the feeding trials. Five weanling pigs, weighing 8.6, 8.6, 9, 9.5 and 9.5 kg. (19, 19, 20, 21 and 21 lbs.) were purchased from a local farm for this experiment.

The three lighter pigs were placed upon a diet of Triplet wheat July 7. The wheat was supplemented by small amounts of tankage, bone meal, alfalfa meal, salt and cod liver oil. At the same time the two heavier pigs were placed on a ration consisting of approximately 40 per cent *A. intermedia* seed and 60 per cent cracked Triplet wheat. This degree of purity was obtained by separating the débris of the wheat plant and other lighter materials by floatation and the small particles by screening. Only an occasional mustard seed remained and, as far as possible, these were removed by hand picking. In addition to the screenings the pigs were given the same supplements as the pigs on the clean Triplet wheat. They were maintained under identical conditions.

On August 14, after 38 days of experimental feeding, the three pigs on Triplet wheat were in excellent condition and weighed 21.4, 21.4 and 23.6 kg. (47, 47 and 52 lbs.). The pigs receiving the screenings which contained *A. intermedia* seed had assumed the appearance typical of early cases of hard liver disease and weighed 11.4 and 12.3 kg. (25 and 27 lbs.). The pig weighing 11.4 kg. was killed. Icterus was absent and microscopically the liver appeared normal. Microscopic examination of fixed sections of the liver, however, revealed the beginning of necrosis of toxic origin. Slides were submitted to different pathologists for d i a g n o s i s. Dr. Stier[10] noted a similarity to the changes in human livers associated with excessive alcoholism, and Dr. Smith[11] diagnosed the condition as beginning necrosis of toxic origin in which some of the cells could already be pronounced necrotic on the basis of karyolysis and absence of nuclei.

On September 6, after 61 days of experimental feeding, the pigs fed Triplet wheat were in excellent condition and weighed 31, 32.7 and 35 kg. (68, 70 and 77 lbs.) The remaining pig on the diet of screenings had lost 1.4 kg. (3 lbs.) in 23 days and weighed 11 kg. (24 lbs.). This pig and the 32.7-kg. control are shown in figure 7. The pig was killed, and while no icterus was present, the liver was much firmer than normal. Microscopic study revealed extensive hepatic destruction.

The three pigs on Triplet wheat continued to gain and, as is shown in figure 8, taken at the end of 110 days of experimental feeding, their appearance was normal. At this time they weighed 51, 52.3 and 53.2 kg. (112, 115 and 117 lbs.). Two were slaughtered October 31 and presented no evidence of hepatic disorder. One pig was placed upon another feeding experiment.

FEEDING TRIALS WITH HORSES

Two equine animals, one a colt estimated to be 6 months of age, the other a 2-year-old, were placed in a box stall and fed wheat screenings, commencing December 16. In addition, a limited amount of alfalfa hay was fed. The amounts consumed by both animals, but not by each, are known. During the first few days, both ate the screenings with relish, consuming as high as 6.4 kg. (14 lbs.) a day, of which one tenth to one fourth consisted of the seed of *A. intermedia*. On the tenth day both went off feed, refusing both alfalfa hay and the screenings. They were gradually brought on feed again with clean rolled oats and alfalfa hay. Limited amounts of screenings, usually not over 0.9 kg. (2 lbs.) per day, were consumed throughout the remainder of the experiment. The colt died February 15, after 61 days of experimental feeding. Icterus was pronounced and the liver was somewhat enlarged. Microscopic examination revealed evidence of both hepatic cell destruction and proliferation.

The horse died May 4, after 138 days of experimental feeding. Before death this animal showed some cerebral symptoms and was difficult to handle. Icterus was noted clinically. Autopsy revealed a hard, cirrhotic liver and extreme icterus throughout.

Another horse, approximately 2 years old, was placed on experimental feeding with screenings which averaged 13.5 per cent *A. intermedia* seed, June 8. As was noted with the other horses, this animal relished the screenings for the first week and then was made ill, after which it consumed the screenings only when other feed was withheld. The first symptoms of cerebral disturbance and icterus were noted July 25, after 47 days of experimental feeding. The horse would lean on the stall or manger or upon an attendant and would stand with the legs crossed. Hemoglobinuria and extreme icterus were noted on July 29. The horse died on August 2 and autopsy revealed an extremely hard, cirrhotic liver and pronounced icterus throughout the entire body. The spleen also appeared to be enlarged and more fibrous than normal, and the kidneys were hemorrhagic.

In the field, clinical cases of walking disease usually are first observed about the time of spring plowing, after winter feeding on screenings or at straw stacks, where the horses would consume small amounts of *A. intermedia* seed with the salvaged grain.

Feeding Trials with Calves

On April 17, a Holstein calf, approximately 3 weeks of age and weighing 39 kg. (86 lbs.), was put on experiment. Its sole ration consisted of 5.67 liters (6 qts.) of warm, fresh, skim milk per day, divided between three feedings, to which was added approximately 100 Gm. (3.3 oz.) of ground wheat screenings rich in *A. intermedia* seed. On May 4, after 18 days on this diet, the calf showed acute abdominal pain and a tendency to bloat. Death followed within a few hours. The liver was definitely hard and cirrhotic and icterus was pronounced throughout the body.

On May 18, another Holstein calf was obtained and given hand-picked *A. intermedia* seed in gelatin capsules. At the beginning of the experiment this calf weighed 48 kg. (105 lbs.) and was approximately 6 weeks of age. It was given approximately 10 Gm. (0.33 oz.) of the pure, whole seed daily for 30 days. It weighed 67 kg. (147 lbs.) on July 7 when it was killed. There was no icterus and no gross evidence of hepatic cirrhosis. The seed used in this trial was obtained from screenings which had not been used previously for feeding, although the seed appeared to be similar to those used in previous feeding experiments.

A third calf, a Jersey weighing 34.5 kg. (76 lbs.) and approximately 5 weeks of age, was given 50 Gm. (1.66 oz.) of ground *A. intermedia* seed from the same lot as was fed the second calf, beginning June 13. This was continued until June 30. When killed July 7, this calf weighed 35.4 kg. (78 lbs.). There was no icterus and no gross evidence of hepatic cirrhosis.

Fig. 9. Control bird and bird fed A. intermedia seed. Both were maintained on a basic diet deficient in vitamin D. Note the comb development in the bird fed the seed. Liver damage did not occur in fowl.

These experiments indicate either that calves vary greatly in their susceptibility to the toxic principle of *A. intermedia* or that different lots of seed vary greatly in the amount of toxic principle present.

Feeding Trials with Chickens

On December 19, twelve male White Leghorns, hatched October 5, 1938, were divided into two groups, one to serve as a control and the other to be fed a ration high in the seed of *A. intermedia.* The birds in the test group weighed 467 to 563 Gm. (15.5 to 18.8 oz.) and averaged 510 Gm. (17 oz.); those in the control group,

440 to 540 Gm. (14.6 to 18 oz.) and averaged 492.5 Gm. (16.4 oz.). The controls were fed a ration used by the division of poultry husbandry. The test birds were first fed equal parts of this ration and screenings rich in *A. intermedia* seed. Later, the proportion of screenings was increased to two thirds.

The birds ate the *A. intermedia* seed with apparent relish. Although the gains of the birds on the screenings were less than those made by the controls, the average weights on February 27 being 1,060 Gm. (2 lbs., 3 oz.) for the birds receiving *A. intermedia* and 1,158 Gm. (2 lbs., 6.6 oz.) for the controls, and 1,193 and 1,502 Gm. (2.5 and 3.1 lbs.), respectively, on March 30. By June 1, the birds in the control pen began to show pronounced leg weakness and pale combs, probably due to the deterioration of the cod liver oil in the ration. The test birds continued to make slow gains and appeared normal. During the month of June, all of the birds in the control pen developed leg weakness and died, while those receiving the screenings rich in *A. intermedia* seed were in good condition and showed abnormal comb development. The contrast in appearance is shown in figure 9. These experiments were conducted in a room devoid of sunlight and illuminated by an electric light. Postmortem examination of the experimental birds failed to reveal any evidence of abnormality. While not favorable to rapid gains and therefore not a desirable feed, these screenings appear to protect against vitamin D deficiency and possibly contain some substance which stimulates comb development. This point will be investigated in future experiments.

FEEDING TRIALS WITH A SHEEP, A RABBIT AND WHITE RATS

A 25-kg. (55-lb.) lamb was placed on a ration consisting of equal parts of the basic ration fed the pigs and wheat screenings rich in *A. intermedia* seed, on December 20. Although it did not relish the seed, considerable amounts were eaten, at times over 200 Gm. (6.6 oz.) in a day. On February 18, it weighed 33 kg. (72 lbs.). The morning of March 31 it was found out of its crate and dead. Extensive subcutaneous hemorrhages in the pectoral and abdominal regions were observed at autopsy, which would indicate an unexplained accident. No evidence of icterus or hepatic cirrhosis was observed. The postmortem weight was 29 kg. (63 lbs.).

A white rabbit, weight 1 kg. (2.2 lbs.), was placed on a diet of one-fourth basic ration fed the swine and three-fourths screenings rich in *A. intermedia* seed. The rabbit ate the seed with relish and has remained in excellent condition. Its weight on September 20 was 3 kg. (6.6 lbs.) and there was no evidence of hepatic changes or icterus.

Five white rats were placed on levels of 10 to 25 per cent ground *A. intermedia* in their ration on December 18, and five more on December 20. While those on the higher levels of screenings made less rapid gains and did not relish their feed, when all were killed and examined on April 4, no evidence of icterus or hepatic cirrhosis was found.

PATHOLOGY*

The most striking pathological change observed in all pigs and horses, and in one of the three calves fed, was an extreme destruction of the hepatic tissues and replacement with fibrous tissues. In very acute cases the liver was swollen; in more chronic cases, it was somewhat shrunken and extremely hard. In one chronic case, adhesions had formed between the lobes of the liver, and the wall of the gall bladder was extremely thick and fibrous. Except in those cases where an unusually severe anemia had developed, icterus was pronounced. Microscopically, the livers of the pigs fed for only short periods revealed a beginning necrosis with some of the cells showing karyolysis and absence of nuclei.

The changes reported by Hilton Smith in these early cases were:

Acute toxic hepatitis with progressive ne-

*The pathology of amsinckiosis will be published at a later date.

crosis. Changes precursory to necrosis of the individual liver cells appear to be: 1) Marked swelling, possibly due to imbibition of water (not characteristic of cloudy swelling as it is understood in America); 2) liquefaction and dissolution of the cytoplasm, beginning peripherally with irregular precipitation of cytoplasmic residue; 3) karyolysis; and 4) loss of cell outline and loss of differential staining. The islands of Glisson show noticeable eosinophilic infiltration but no cirrhosis. A special stain for fat showed complete absence of degeneration of this type.

The livers of pigs which had been allowed to continue on experiment until death revealed an extreme hypertrophic cirrhosis with hyperplastic and toxic changes in the remaining parenchyma. The lobular architecture was obliterated and the connective tissue exceeded the parenchyma in amount. The parenchymal cells often were greatly enlarged and some contained several nuclei, as if from confluence of several cells. In some of the sections examined, not more than four or five normal hepatic cells appeared in a microscopic field. The spleen also was involved and in many of the acute cases was enlarged, while in the chronic cases it appeared to be somewhat fibrotic. The kidneys were hemorrhagic in some horses and the bladder filled with blood-stained urine.

A. INTERMEDIA

A. intermedia has not previously been described as a poisonous plant, although the plant, shown in figure 10, is commonly found in western grain fields. Lawrence,[12] in 1922, investigated walking disease in eastern Oregon and wrote:

> There is a general belief that the tarweed, *A. intermedia*, causes the walking dease among horses. Whether this plant is responsible for the sickness has never been definitely established. Until the tarweed is definitely determined to cause the sickness, no recommendation should be made.

The poisonous plants committee of the Commonwealth of Australia[13] suspected

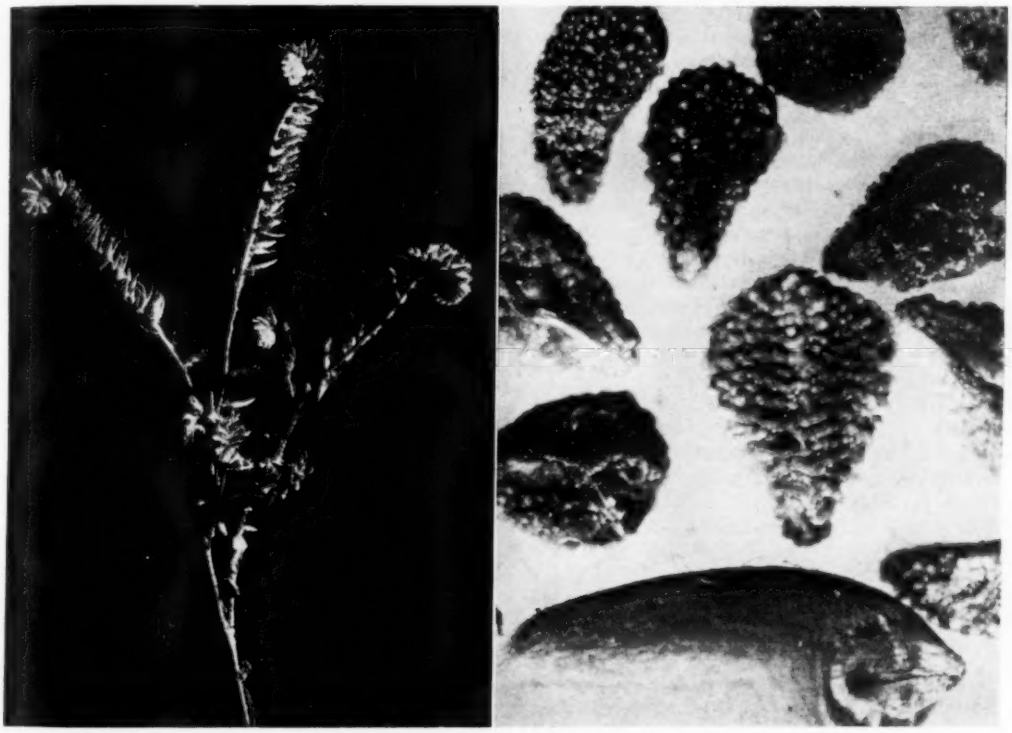

Fig. 10 (left). Branch of A. intermedia from Walla Walla. This is the most common weed in grain fields in many regions and grows profusely along the roadsides. Fig. 11 (right). A. intermedia seed with one grain of triplet wheat (x 12).

that this plant is toxic and had tests conducted on sheep, but found it nontoxic. Pammel[14] names *A. intermedia* as one of a number of plants which produce mechanical injuries. The following description of the plant is given by Muenscher[15]:

Amsinckia intermedia, Fisch. and Mey.

Other names: Tarweed, buckthorn-weed, finger-weed, fiddle-necks, fireweed, yellow burnweed, yellow forget-me-not.

Annual; reproducing by seeds. Grain fields, gardens, orchards, meadows and waste places.

Native to the Pacific Coast; introduced locally in the central and eastern states, from May to July.

Stems erect or with spreading branches, 3-8 dm. (11.8-27.5 in.) long, covered with stiff white bristly hairs. Leaves alternate, lanceolate to linear, rather thick and covered with short bristly hairs. Flowers perfect, regular, in a dense, one-sided coiled, leafy-bracted raceme which becomes much elongated in fruit; calyx with 5 linear, acute, very densely bristly lobes; corolla salverform, 5-lobed, yellow, 5-6 mm. (0.19-0.23 in.) long; stamens 5, separate, inserted on the corolla-tube; ovary deeply 4-lobed, in fruit separating into 2 or 4 seed-like nutlets which are attached above the base. Nutlets about 2.5 mm. (.098 in.) long, ovoid, angular, apex curved, with a scar near the base, somewhat winged on one angle, unarmed but roughened by wrinkles, gray to dark brown or nearly black. A variable species represented by several forms which are sometimes separated as species.

These seeds, which are enlarged 12 times in figure 11, are a common impurity in grain and constitute a considerable percentage of the heavy screenings in some regions.

Pammel[14] gives fireweed as a synonym of *A. tesselata* and buckthorn as a synonym of *A. intermedia*.

While growing most profusely in certain of the winter-wheat areas of Washington, Oregon and Idaho, it has a much wider distribution. As early as 1890, Hilgard[16] reported *A. intermedia* as a troublesome weed in California.

Muenscher[17] does not include *A. intermedia* in his recent text on poisonous plants and mentions only one member of the borage family, *Echium vulgare* L., which he describes as producing a dermatitis accompanied by severe inflammation and itching.

In his monograph on "Flora of Southeastern Washington and of Adjacent Idaho," St. John[18] listed six species of Amsinckia. In addition to *A. intermedia*, which he described under the name of *A. lycopsoides* Lehm., with the observation that, "The genus is in need of a careful monographic revision," he described *A. densirugosa*, *A. Hendersonii*, *A. micrantha*, *A. retrorsa*, and *A. washingtoniensis*. The variability of these species and the difficulty in differentiating between some of these species, especially where only the seed is obtained, has been confirmed by E. F. Gaines of the State College of Washington.

Therefore, it is possible that the seed of species of Amsinckia other than *A. intermedia* may have been included in some of these feeding experiments.

DISCUSSION AND SUMMARY

The toxicity of the seeds of the yellow burweed or yellow tarweed, *Amsinckia intermedia*, for horses, swine and cattle has been demonstrated. This apparently is the first time any member of the family Boraginaceae has been shown to be poisonous. The seeds of *A. intermedia* have been shown to be responsible for enzoötic hepatic cirrhosis, known as walking disease of horses and hard liver disease of swine and cattle, as it occurs in certain regions of the Pacific Northwest. Rabbits, rats and chickens appear to be immune and our data to date show sheep either to be immune or highly resistant. No treatment for enzoötic hepatic cirrhosis is suggested. Prevention consists of avoiding feeds containing the seed of *A. intermedia*.

The data indicate that considerable amounts of the seed must be fed before pathological changes are produced, and the epizoötiology of the disease indicates that the seeds vary in toxicity during certain years. There also is the possibility that the seeds of this plant, grown in other regions with different soil and climatic conditions, may be less toxic. The maturity of the plants when the grain is harvested may also influence the toxicity of the seed and it is possible that there may

be more than one variety of the plant in the areas studied and that the seeds from these varieties may differ greatly in toxicity.

It is probable that poisoning by *A. intermedia* has escaped recognition in those areas in which only a few farms are sufficiently infested with this plant to make the grain or screenings capable of producing fatal hepatic cirrhosis. Sublethal poisoning of swine by the seeds of this plant results in failure to grow and loss of condition, which easily could be attributed to other causes, especially since mildly affected swine present an appearance commonly associated with parasitism.

The exceedingly irritant nature of the standing plant makes it appear improbable that the plant would be eaten by live stock, although cattle are said to graze sometimes upon the very young plants. The seeds, however, after passing through the harvester, are not irritant and possess a pleasant, nut-like flavor.

The present plan of limitation of wheat acreage, which includes some of the wheat land remaining idle, is favorable for the growth of this weed and it is possible that it may become sufficiently established to cause trouble in localities which heretofore have escaped.

The reports of other workers indicate poisoning by members of the genus Senecio to be responsible for enzoötic hepatic cirrhosis over a larger portion of the United States than poisoning by the seeds of *A. intermedia*. The pathological changes in the liver produced by *Senecio Reddellii* and the seed of *A. intermedia* are similar.

Alcoholic extracts of the seed contain the toxic principle of *A. intermedia*. Further studies are being made to ascertain its chemical nature.

ACKNOWLEDGMENTS

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Controlling Diseases and Parasites in Garbage-Fed Hogs*

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DURING the past three decades, the feeding of garbage to hogs has developed into an extensive business. It is recognized as the only economical way to dispose of this commodity. It can be, and usually is, a profitable venture, while any other method of disposal is a failure from an economic standpoint and, in some instances, actually becomes a health menace as well.

Inasmuch as the material in this report is compiled from the actual experience of the writer, it should be mentioned that this experience has been gathered only in Colorado and California.

Denver, Colo., is divided into several districts and one hog raiser collects all of the garbage in his district. Most of the ranches are located in a rather isolated section. This system has worked out to the benefit of the citizens and the profit of the feeders.

In southern California, conditions are almost ideal climatically for the feeding of garbage to hogs. In addition to mild winters, moderate rainfall and sandy soil, there is also a demand for fertilizer, which is an important by-product of garbage.

Garbage feeding presents a multitude of problems not common to farms on which hogs are fed grain and pasture crops. It is customary to build houses and pens on a more or less permanent basis. Feed floors, water troughs and shed floors are of concrete. As the owner must keep construction costs down, only enough housing is provided to take care of the hogs actually on hand. There is a tendency to overcrowd the pens, thereby increasing the hazards of intestinal and respiratory diseases.

Garbage contracts are usually let for five to ten years. Therefore, hogs are kept in these pens constantly during this period and the soil is consequently contaminated with whatever pathogenic organisms are in the excrement.

A system of sanitation must be maintained, including regular cleaning of feed floors, water troughs, pens and sheds. Breeding places and roosts of flies must be sprayed, and the roadways kept free of garbage which spills from the feed trucks. Concrete wallows should be provided for hot days. These require frequent cleaning.

In speaking of industries in southern California, no particular emphasis is placed on modesty. Perhaps the writer may be excused, then, for saying that at Fontana, Calif., we have the largest hog ranch in the world. Since it was opened in 1921, hogs have been running over the same ground. Since January 1925, except for a year, the writer has been employed as veterinarian.

Obviously, an article on garbage feeding would be incomplete without some reference to this farm. Likewise, a history of disease problems encountered here would carry with it reference to most of the diseases, with the exception of swine erysipelas, found in hogs in the United States.

This ranch has a population of 60,000, including all pigs. All are raised here. A purebred herd is maintained to produce boars. There are three separate breeding herds of some 3,000 sows and gilts and 100 boars each. Inbreeding is prevented by

(*Continued from preceding page*)

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*Presented before the Section on General Practice at the 76th annual meeting of the A.V.M.A., Memphis, Tenn., August 28 to September 1, 1939.

purchasing outside boars for the purebred herd and changing the strain for the breeding herds. From 1,000 to 1,200 sows are bred each month. All hogs are fattened here, except some purebred boars and gilts.

The daily feed supply consists of 400 tons of garbage shipped from Los Angeles, about 45 tons of grain and four to five tons of green feed—or, in winter, dry alfalfa.

In 1938, 55,132 pigs were weaned. In the same year 54,947 hogs, weighing 11,469,751 lbs., were marketed. In 1939, fertilizer sold amounted to over 26,000 tons. At 8 cents per pound for the hogs sold and $4.00 per ton for the fertilizer, we have here a million dollar business.

If one considers that we use only part of the garbage from one of the larger cities of the country, it can readily be seen that garbage feeding in the United States might well be a 50 million dollar industry.

As graduates of institutions which are supported by federal and state appropriations which, in turn, are inspired by agricultural and livestock interests, we must dedicate ourselves, in part at least, to the control of diseases of animals which are utilized in the production of our food. Of particular concern are those diseases affecting lower animals to which human beings are also susceptible. Of greater economic importance, no doubt, are diseases that affect only animals, or human beings rarely, but which, if not controlled, might seriously threaten our meat supply.

TRICHINOSIS

In the recommendations of regulations calculated to be a boon to public health, let us exercise care that an industry may not be jeopardized. In recent years there has been produced certain evidence that the feeding of garbage to hogs has contributed largely to the increased incidence of trichinosis in man. In California, the available information has been greatly exaggerated by a few individuals who are interested in selling incinerators to cities. Their propaganda is difficult to combat except by publication of the fact that trichinosis exists also in farm-fed hogs. Such a countercharge, however, would serve only to bring hog raising into disrepute and reduce the consumption of pork.

It is generally accepted that the consumption of raw pork scraps constitutes the principal channel by which trichinosis is transmitted, whether it be in human beings or swine. In grain-fed hogs the disease is found much less frequently. As a matter of common knowledge, a large percentage of farm-raised hogs are fed garbage from the home. In view of the number of farm-fed hogs marketed each year, there might conceivably be more actual cases of trichinosis in farm-fed hogs than in those fed garbage as the basic ration, even though the percentage of cases be higher in the latter.

The writer observed in Colorado and California that the cooking of garbage greatly reduces its feeding value. This was especially noticeable in sows and young pigs. Most of the pigs were runts. It is highly probable that many garbage feeders in California would quit the business if sterilization of garbage were enforced, because the occupation would cease to be profitable.

Obviously, any program pointing toward eradication of trichinosis that fails to take into account the widespread presence of this disease in farm-fed hogs would be destined to failure. Therefore, before we go on record as favoring the sterilization of garbage that is to be fed to hogs, it seems advisable for us to ascertain how much damage would be done to the industry in question as well as how inadequate the efforts would be unless they include farm-fed in addition to garbage-fed hogs.

Several veterinarians in this country have studied this problem in detail and at times issue statements on the subject to the popular press. Editors often distort the information given them and, in turn, their reports are frequently used by unscrupulous persons in a way that tends to curtail hog raising and pork consumption, all of which is detrimental to any coöperative program intended to eradicate trichinosis.

HOG CHOLERA

Hog cholera is of course the major disease to combat in garbage-fed hogs, as it is on other farms where hog production is of importance. Vaccination of baby pigs

has long been a controversial subject. On grain ranches vaccination is frequently delayed until after the pigs are weaned. This is a satisfactory plan but it can not be followed on large garbage-feeding ranches due to the fact that a certain percentage of pigs will contract cholera before they are old enough or large enough to be weaned. Then, baby-pig vaccination is a necessity. In the early 1920's, around Denver, Colo., it was common experience among the garbage feeders to have "breaks" some months after vaccination. Large numbers of hogs would die. These feeders usually did their own vaccinating or employed a neighbor somewhat proficient in the art. They would seldom, if ever, call a veterinarian.

Unofficial observations strongly indicated that most of their losses were due to hog cholera. Adequate and lasting immunity had not been developed. This implies no criticism of methods or products. It might be better described as a phase in the knowledge of production and administration of serum and virus, in both of which we have progressed. In 1925, when the writer first took over the work at the Fontana farm, pigs were being inoculated with 2 cc. (30 minims) of virus and 25 to 30 cc. (0.85 to 1 oz.) of serum at 6 weeks of age.

The virus was shipped across several states under rather irregular conditions of refrigeration. Cans of ice water were used to keep the virus cool in the field. In short, the need of keeping virus at a low temperature was recognized and an attempt was made to follow this requirement. Also, the dose of virus was increased to 2.5 cc. (35 minims), yet a cholera break in the pigs occurred in October and November. Several thousand hogs were revaccinated. In 1926, single vaccination was tried again and, late in the year, cholera was in evidence once more. After revaccination, it was decided to try two inoculations—one at four to five weeks and one after three months. This plan proved effective in controlling cholera but it had the disadvantage of added expense as well as a second handling of the pigs.

In 1932, upon the recommendation of a representative of the federal bureau of animal industry, another attempt was made to employ only one inoculation. This time the break was delayed but, early in 1933, after the writer had left this farm, cholera appeared and a severe loss was suffered during that year. The double method was again resorted to, with good results.

In 1932 and thereafter, an excellent method of shipping the virus from the factory was established. It was packed in an insulated container with dry ice in a box at the top, and a thermometer was inserted to record temperature variations. Shipment was made direct from factory to farm, the virus being enroute about three days. An electric refrigerator was installed for storage. Ice was used in an insulated container when virus was taken to the field. This method of handling virus is believed to be beyond criticism.

When the writer returned to this farm in June 1934, it was decided to try the single inoculation in pigs raised from one breeding herd, with 5 cc. (0.17 oz.) of virus as the dosage. This procedure was followed for about a year, during which over 2,000 pigs were given a test dose of 1 cc. (15 minims) of virus at 4 to 6 months of age. The results were so favorable, only two pigs failing to stand the test, that the method was used on all pigs in the spring of 1935. The tests in these pigs were almost entirely satisfactory.

A change of serum and virus supply was made for 1936. The virus was apparently not carefully shipped or lacked the high degree of virulence required to produce lasting immunity. That fall there was another outbreak of cholera and revaccination was employed on some 12,000 to 15,000 animals.

Beginning in 1937, the previously described method of shipping was used. Since that time, cholera has apparently been satisfactorily controlled by the administration of 5 cc. of virus and 22 to 32 cc. (0.7 to 1.07 oz.) of serum, depending upon the size of the pigs, at an average age of 5 to 6 weeks. No "breaks" have been observed and the pigs tested have with a few exceptions remained healthy. This is the way that hog cholera has been controlled on the largest hog ranch in the world. The virus is delivered in 500-cc. (16.6-oz.) bottles. A filler and rubber tube connects this bottle

to a 10-cc. (0.33-oz.) syringe. As pigs are vaccinated at the rate of 250 to 300 per hour, the virus does not get warm and is not unduly exposed to light.

Last year, the county veterinarian of Orange county, Calif., called on the writer for a consultation. The garbage feeders in his county were experiencing losses from cholera. It seems probable that he persuaded them to adopt the same methods used by the writer and recommended to him. As no word has been received from him recently, it is assumed that he was able to conquer his problem.

In the description of this plan of cholera control, for which no claim of superiority is made, details have purposely been stressed. On account of the diversity of opinions and experiences among veterinarians, it seemed likely that a definite record of baby-pig vaccination in rather large numbers over a period of years, followed by a more than average severe field exposure, plus several thousand inoculation exposures with results that were quite satisfactory, might serve as a guide to those confronted with similar problems. Pigs immunized in the manner described are kept on the ranch for many years as breeding stock. There is no evidence of loss of immunity.

In recent years a great deal of work has been done on the production of a vaccine for hog cholera. One of these vaccines, developed by Wm. H. Boynton, has been used by the writer. In a recent experiment it appears to have produced a high degree of immunity.

Foot-and-Mouth Disease

In 1924, during the severe outbreak of foot-and-mouth disease in California, the disease made its appearance at this ranch. Some effort was made to prevent its introduction by cooking the garbage, which, however, did not exclude the malady but which did add definite evidence to our conviction that cooked garbage is not a satisfactory basic feed for small pigs. The eradication of this disease was handled in the usual way and this, fortunately, checked its spread, limiting the loss to a few thousand head of hogs.

Vesicular Exanthema

In 1935, there appeared in several hog ranches in California a disease so closely resembling foot-and-mouth disease in hogs that differentiation was possible only through animal inoculation. This was vesicular exanthema and was present in the hogs at the Fontana farm for some months. A quarantine was established and no visibly infected hogs were permitted to leave the ranch. No treatment was given. The result was a gradual clearing up and, finally, a complete disappearance of the trouble.

Bronchitis

Bronchitis of young suckling pigs occurred about in 1926 in one of the three breeding units. It was observed that quite a number of the animals developed a serious cough when they were about 5 weeks of age. This disease was usually confined more to the bronchi and anterior lobes of the lungs. The immediate loss from deaths was light but the pigs lost weight and condition and became subject to increased loss later by more readily contracting respiratory diseases. All of them took longer to reach marketing weight, which meant more feed and an additional expense.

In an attempt to determine the cause and a preventive treatment, a number of veterinarians were consulted. Various biological products were given the pigs, usually two doses at the ages of 2 and 3 weeks, in an attempt to prevent their contracting the infection. Hemorrhagic septicemia bacterin and aggressins, autogenous bacterins and mixed infection bacterin were used. In the writer's hands these products had no effect. Then, concrete was used in the lots so that they could be washed and disinfected, but with no beneficial results. Attention is called to the fact that there were three breeding units fed and housed under almost identical conditions, yet this bronchitis affected pigs in only one unit, although at times pigs in another unit were less than 100 yds. distant. Thirty to 40 per cent of the pigs in the unit were affected in varying degrees. Finally, as in the case of the

vesicular exanthema, natural forces came to our rescue. About 1½ to 2 years after its appearance the bronchitis disappeared.

PNEUMONIA AND GASTROENTERITIS

Three or four times in the past ten years we were confronted by outbreaks of an acute pneumonia that caused the loss of several hundred pigs, commonly between the ages of 3½ to 4½ months. It was usually not difficult to isolate *Pasteurella suisepticus*, although on some occasions *Salmonella suipestifer* seemed to be the causative agent. Neither commercial nor autogenous bacterins or aggressins proved beneficial in the prevention or control of this disease. Acute gastroenteritis is another disease that causes the garbage feeder a great deal of concern. Both of these diseases are of rather frequent occurrence and the underlying cause is usually insufficient sanitation.

Proprietors of garbage-feeding ranches are in the business for profit. It is important to keep the investment as well as operating costs at a minimum. In California the long summer with no rain permits quick drying of fecal matter, which then mixes with the layers of dust in pens where hogs are kept. Water in large amounts is needed on inland farms for drinking and for the purpose of cooling. Concrete wallows are expensive. It is much cheaper to run water into a low place for a wallow. It is also expensive to clean pens frequently and sprinkle them to keep down dust. These factors make the summer in California the time when heaviest losses occur from the acute type of pneumonia and enteritis. When such conditions are coupled with overcrowding, a rapid spread of diseases may be expected.

It is true that rather often we find pigs thriving even though sanitary conditions are almost deplorable. However, the writer believes that where good sanitation is the general practice and regularly maintained, pneumonia and enteritis can be controlled.

When these diseases are observed, the dietary is altered and the garbage taken away until the acute attack subsides. Bran mashes and green feeds are given. Following these acute outbreaks, there are always a certain number of animals which develop chronic symptoms. They are sorted out and placed in pens by themselves, where with access to soft feed a fair percentage recover. Quite a number of these break at a later period, others are difficult to fatten, and some are shipped to market only to be condemned.

ANEMIA

Anemia may assume serious proportions in young pigs that are farrowed on concrete or wood floors. Many garbage ranches follow this method; hence, it is well to give this disease particular consideration. It is rather easily recognized. In pigs that have been fat, a wrinkling of the skin is noted and a yellowish discoloration appears on the skin of the abdomen. A simple way to prevent this malady is to throw a shovelful of dirt into the farrowing pen every two or three days at the time that the pigs are just a few days of age. In most instances they will find the necessary ingredients in the dirt to prevent anemia. The writer considers this plan much more practical than the application of chemicals to the sow's udder. There is hardly need to repeat that the vaccination of pigs suffering from anemia is likely to be accompanied by numerous deaths.

MANGE AND LICE

We may consider mange and lice in hogs in one discussion, as the application of oil spray seems to be effective for both. Both of these parasites prefer the lower part of the hog's body and the legs; consequently, either may prevail for quite a while without the owner's observing it and may be affecting a large number of hogs. Grease in the garbage is an effective lice repellent in the hogs having access to feed floors where the garbage is fed freely. They are found more often on sows that have been fed less abundantly on garbage. From the sows they go to the pigs, where they are easily found at the time the animals are vaccinated. Whenever the pigs are badly infested, they are sprayed with oil at weaning time. If the infestation is slight, the sows are sprayed when the pigs are weaned. Two or three applications of

the oil at ten-day intervals are usually required to eradicate mange in a herd. If the attendant is taught to observe closely for any skin scaliness in the pigs, the disease can be limited.

Garbage is apparently an efficient vermifuge. Worms do not stay long in the intestine of a hog fed garbage and, therefore, need not be considered in this discussion.

Acute Enteritis

Acute enteritis of pigs 2 or 3 days of age is found commonly on garbage ranches. It is usually more severe in pigs from gilts. It has been said that this is a nutritional disturbance but to the present the writer has found no satisfactory explanation of its cause and no effective means of prevention.

Location and Planning of a Garbage-Feeding Ranch

There are certain factors to be considered in the location and planning of a garbage ranch. It should be far enough from dwellings to prevent its being tabbed a nuisance. The ground should be sandy and sloping. The feed floors should be placed parallel to the natural fall of the ground, not across or at right angles. This permits the drainage to flow away, instead of its being stopped back of the feed floors, causing a muddy approach, or in front, causing a muddy road. There should be a concrete gutter in front of the feed floor into which drainage from the feed floor, the water trough and the concrete wallow will flow. This gutter should be strong enough and wide enough to support a wagon or truck wheel—whichever is used for feeding. Also required are a concrete platform for washing wagons and trucks and a boiler to provide steam and hot water. Some thought should be given to the utilization of the drainage as well as the fertilizer. The former may be diluted with irrigation water and used on vineyards or orchards, where it would furnish valuable fertilization.

Pork from hogs fed exclusively on garbage is not equal in quality to that from grain-fed hogs. It takes longer to harden in the cooler and will not stay as firm at the butcher shop. In hogs finished 30 or 40 days on grain mixtures, the quality is greatly improved. Garbage feeders should adopt this method for their own benefit and protection.

Cities and army posts and other camps that are sources of garbage in large amounts should be more concerned than they have been with regard to how the garbage is handled at the feeding establishment. Longtime rather than annual contracts should be let in order to encourage investment sufficient to insure reasonable sanitary methods.

No one who has made a study of the feeding of garbage to hogs doubts its practical or economic advantage over any other method of disposal. This is particularly true in areas where moderate climatic conditions prevail. Garbage feeding has long been in disrepute, but there is no logical reason that this should be the case. Where a contract is let for only one year, as is the practice at army posts, no one can expect a feeder to provide a properly equipped place when the chances are equal that someone will outbid him the ensuing year.

A satisfactory sanitary condition can be maintained on garbage-feeding ranches, provided that responsible individuals have the contracts. They will bid on contracts if the opportunity for profit seems to justify the investment.

Perhaps veterinarians, as individuals or in groups, should consider making recommendations to civic authorities and should have information available for prospective garbage feeders.

Experiments carried out in France have shown that the pH of the soil is responsible for the appearance of swine erysipelas on certain farms year after year.

Correction: In the article entitled "The Control of Animal Diseases in the Philippines," published in the September 1939 issue, the word "blackleg" (column 1, line 4, page 298) should have been "blackhead."

Diseases Incident to the Fattening of Lambs*

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DISEASES causing the heaviest death losses among range-bred lambs while they are being fattened for market may be divided into two groups: First, those of an infectious nature, contracted as a result of lowered resistance during transportation from the range to the feed pen and, second, those due to errors in diet. Among those recognized as shipping diseases are paratyphoid dysentery, hemorrhagic septicemia, contagious ecthyma and nonspecific pneumonia. Under diseases caused by errors in diet are coccidiosis, overeating, indigestion and nonspecific diarrhea. Internal and external parasites can not be classed with either of the foregoing groups but should be mentioned as feed-lot diseases.

The vast areas of the western range states comprise the breeding ground of the millions of lambs which are transported to the feed pens annually. The long distance on the trails to the loading points on the railways, and the subsequent hours on trains, especially in stormy weather, subject the lambs to infections through fatigue and exposure. The practice of requiring a 12- to 24-hour fast period without feed or water before weighing the lambs to be sent to the consignee, and immediately loading them for a 36-hour ride on the cars, should be discouraged. Shrinkage figured on a percentage basis should be substituted and shorter periods between rest and feeding should be encouraged. Every effort should be made to place the lambs in the feed pen in a strong, vigorous condition.

While Colorado is considered by many to be a range state, approximately 2 million lambs are fattened there annually. In spite of the geographical proximity of the lamb breeder to the feeder under our western conditions, with the high incidence of shipping diseases incurred, it is not unreasonable to assume that feeder lambs shipped to the more remote middle western and eastern states would, in proportion, suffer from a higher frequency and severity of these conditions. Furthermore, the susceptibility of feeder lambs to shipping diseases is common, regardless of their destination, while those diseases resulting from errors in diet vary according to the feeding methods followed and types of feeds used in the various sections of the country.

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PARATYPHOID DYSENTERY

Paratyphoid dysentery has been recognized and reported by the Colorado Experiment Station and, in all of the outbreaks studied, serious delays in shipping with poor feeding accommodations seemed to be the predisposing factors.

The etiological agent of the disease is *Salmonella aertrycke*. Experiments conducted by the Colorado Experiment Station showed that both fasting and the presence of the organism were necessary to produce the disease. Animals fasted for as little as 24 hours and given 30 cc. (1 oz.) of bouillon culture sickened and died, while lambs which were allowed free access to alfalfa hay and given a like amount of culture lived and suffered no illness.

Symptoms.—There is dullness and loss of appetite and diarrhea which occasionally becomes bloody. Some lambs die quickly, showing only slight scours; others scour profusely and recover. The disease appears soon after the arrival of the lambs in the pens and terminates in ten days to two weeks. Paratyphoid dysentery is differentiated clinically from coccidial dysentery, in lambs arriving directly from the range, by its early appearance, while coccidial dysentery usually appears not before the 18th day after arrival.

Treatment.—Separation of the sick animals from the flock is essential, and the healthy animals must be given wide range

to prevent the spread of the disease. This should be done in spite of the fact that animals having access to feed are not subject to the disease. The sick animals should receive a dose of mineral oil and have access to a light diet of alfalfa or clover hay, oats and bran.

Prevention.—Since it has been proved that long fast periods are necessary for the development and growth of the organism and that animals fed regularly withstand heavy doses of the culture, prevention seems to be only a matter of frequent rest and feed periods during transportation.

HEMORRHAGIC SEPTICEMIA

Hemorrhagic septicemia, like all of the shipping diseases, varies in severity from year to year. Improved railroad-shipping facilities have aided in controlling the outbreaks except in seasons when severe storms occur during the time the lambs are being moved into the feed pens. During certain years, whether it was the condition of the lambs or the character of the weather, this disease took a heavy toll from the feeders.

Cause.—The *Pasteurella oviseptica* is recognized as the organism responsible for this disease. The malady is not considered to be highly contagious and occurs only under conditions that render the animals susceptible. The organisms have been shown to exist in the upper air passages of normal animals and, as in other similar diseases, may multiply and produce serious disturbances when the animals are weakened from hunger and fatigue or any other cause.

Symptoms.—The disease usually appears within seven to ten days after arrival in the pens, with a large percentage of the affected animals showing symptoms at the same time. Those with the acute form often die within 24 hours. In the slow-developing cases, a true pneumonia develops which may cause death in a few days to several weeks. At first, the animals are dejected. The head may hang and the ears droop; there is coughing and sneezing, with a mucopurulent discharge from the eyes and nose; food is usually refused and they appear gaunt. As the disease progresses, breathing becomes more rapid and frequently labored; the temperature often reaches 107° F.

Postmortem Findings.—In cases where death occurs in the first few days, numerous subserous hemorrhages are found, particularly under the pleura, along the ribs, and on the heart. The pleural and pericardial cavities contain considerable straw-colored fluid. The lymph glands are swollen and dark red. The mucous membranes of the entire respiratory tract are congested. In the chronic form, lobar pneumonia is recognized, with the lobes showing a mottled appearance. The lobes may be adherent and the lung fastened to the ribs by a fibrinous pleuritis.

Diagnosis.—Because numerous acute diseases will cause the development of hemorrhages visible on postmortem examination and because the lesions in hemorrhagic septicemia vary greatly, according to the length of time the disease has existed, diagnosis can be established only by the isolation of the organism from the blood or internal organs.

Treatment.—Segregation of the sick animals and giving them the best possible care are the sole recommendations for combating this disease. The writer has found no medicinal agent to be of value. The use of bacterins during an outbreak seems only to aggravate the condition.

CONTAGIOUS ECTHYMA (= SORE MOUTH)

Contagious ecthyma, or sore mouth, is probably the most common of the feed-lot diseases. It is present to some degree in nearly every flock of lambs brought to the feed lots. It appears usually during the first ten days and may exist for two or three weeks, during which time serious losses in weight may occur, owing to the inability of the animals to consume the proper amount of feed and water. The mortality from this disease in its uncomplicated form may not be high, but the economic loss from shrinkage may be heavy. It is caused by a filtrable virus.

Symptoms.—Small vesicles appear on both the inside and outside of the lips and on the gums. Sometimes, the tongue is also

affected. The vesicle soon ripens into a pustule and ruptures. Later, the raw surface is covered with a thick, grayish brown scab. The affection may be confined to the commissures of the lips or it may extend over the entire lips and nose, making it almost impossible for the animal to eat. In some instances, lesions develop in the internal organs and usually prove fatal. Laryngeal ulcers and necrotic pneumonia, as complications following sore mouth, are often the cause of heavy death losses. Ulcers often develop in the rumen and reticulum and may extend even into the entire intestinal tract. In a few instances, the liver is studded with small, necrotic areas. In most of these complications, *Actinomyces necrophorus* will be found as a secondary invader.

Treatment.—The vaccine made from scabs taken from typical cases has proved very satisfactory. Its use in flocks already showing the infection helps immeasurably to prevent serious complications that cause heavy losses and also shortens the time of recovery in the lesions on the lips. The vaccine is applied to scratches on the skin, usually on the inner surface of the thigh, in much the same manner as smallpox vaccine is applied in the human. Other treatment consists of the segregation of the badly affected animals in order that they can be given special care. The removal of the scabs and applications of iodine and glycerin or potassium permanganate solution are helpful. Easily assimilated food and fresh water should be available at all times.

NONSPECIFIC PNEUMONIA

A true pneumonia from which no specific organism can be isolated is encountered at times. Occasionally, it reaches serious proportions. This form of pneumonia is usually the result of some predisposing cause, such as severe chilling, excessive dust or gas, and fatigue during transportation. A large percentage of the smaller and weaker lambs are included among those affected.

Symptoms.—The symptoms and course of the disease are similar to those described for hemorrhagic septicemia.

Postmortem Findings.—Postmortem examination usually shows one or both lungs to be severely congested or consolidated and mottled. The fibrinous pleuritis and pericarditis recognized in hemorrhagic septicemia are absent.

Treatment.—Segregation and good nursing constitute the treatment.

Diseases Caused by Errors in Diet

COCCIDIAL DYSENTERY (= COCCIDIOSIS)

During the past few years, coccidiosis has been one of the most serious of the feed-lot diseases, often taking a toll of 10 to 15 per cent. It is classed by some as a shipping disease, but recent studies showed that proper management and diet during the first three weeks in the feed lot have considerable bearing in the control of the disease. It is possible to demonstrate the presence of coccidia in practically all lots of feeder lambs.

Drs. Deem and Thorp of the Colorado Experiment Station showed that the number of coccidia increase during the first two weeks, remain stationary for about three weeks and then decline. These experiments were conducted in lambs showing no clinical evidence of the disease.

There is little doubt that the sudden change from the open range with natural grass feed to the close confinement of the feed pens, with a concentrated and heavier diet, is the predisposing cause of the disease. Lambs held in close confinement, especially when improperly bedded, are subject to heavy infection from their own droppings. A protozoan parasite, *Eimeria arloingi*, taken in through the mouth and developing especially in the large intestines, is the etiological agent.

Symptoms.—In lambs received at the feed pens directly from the range, the majority of outbreaks occur about the 18th to the 21st day. Scours occurs in a large number at the same time. Often, a bloody diarrhea develops and the animal shows great depression and loss of appetite. It walks with a stiff and arched back, the tail is rigidly elevated and tenesmus exists. The bloody diarrhea continues for several days followed by emaciation and death. The

presence of large numbers of coccidia under microscopic examination of unconcentrated fecal samples confirms the diagnosis.

Treatment.—The sick animals should be segregated each day and the healthy animals given free access to a wide range. Avoid heavy feeds, such as barley and corn, entirely and give only light feeds of oats and bran, and choice hay. Avoid all chance of possible reinfection by providing clean water and changing the quarters frequently. Where wide range is not possible, pens should be deeply bedded and cleaned often. Inclement weather seriously aggravates this condition and special attention is necessary to prevent a high mortality at such times.

The sick animals should be given a liberal dose of mineral oil. Bismuth subnitrate and tannic acid also have been used with some success. The results obtained from the administration of sulfanilamide in similar protozoan infections in dogs, and also in the human, suggest its use in this condition.

Allowing wide range during the day with deeply bedded pens at night and strict avoidance of heavy diet during the first three weeks are important precautionary measures.

OVEREATING

As a result of better breeding stock on the ranges, larger lambs are being produced, many of them weighing 32 kg. (70 lbs.) or more upon arrival at the feed pens. In order to fatten these heavy lambs, and yet not be penalized for having overweight lambs on the market, feeders have attempted to shorten the length of the feeding period by forcing the concentrates as rapidly as possible. This has resulted in a condition known as overeating. Overeating, as the name might imply, does not mean a single engorgement of grain, but is rather the product of the continued consumption of more concentrates than can be properly digested and assimilated. It is a specific entity with characteristic symptoms and pathology. It probably causes a higher death loss in the feed pens of Colorado than all of the other diseases combined.

Cause.—The exact cause of this condition is not yet understood. Lambs in different feed lots will "break" under different amounts of grain, yet there seems to be a fairly definite ratio between the number of days on feed and the amount of grain which can be safely fed. Lambs appear to acquire a toleration for an increase in the amount of concentrates fed but any amount given above this point of toleration will often be accompanied with disastrous results.

Drs. Newsom, Cross and Thorp of the Colorado station showed that filtrates made from the intestinal contents of lambs dead from overeating are toxic when injected into laboratory animals. The filtrates are also toxic when injected into sheep but do not prove toxic when given by the mouth or even when injected in large quantities directly into the duodenum. The neutralizing effects of antitoxins upon these filtrates showed in every instance that *Clostridium perfringens*, type D (*Bacillus ovitoxicus*, type D), and lamb dysentery (type B) neutralized the filtrate, while *Cl. perfringens*, type A (*B. welchii*, type A), and *Cl. paludis* (type C) did not. The factor, then, that permits the toxin to permeate the intestinal walls and cause death has not been definitely determined. It is fully understood, however, that withholding the grain ration always stops the loss immediately.

Symptoms.—The affected lambs are usually the largest, fattest and most vigorous in the lot. In most instances, the affected animals develop cerebral symptoms. They throw the head back (= opisthotonos), turn in circles, or push against objects, such as a feed rack or a corner. In the early stages of an outbreak, the affected animals may go down and lie for days with the head thrown back and keep up an almost constant movement of the legs, while those developing the condition when on a heavy grain ration usually die within a few hours and, in some instances, within a few minutes. Scours is almost a constant factor, evidenced sometimes just before death. Few recover.

Postmortem Findings.—In acute cases the intestinal wall is covered with large, blotchy, subserous hemorrhages. Hemorrhages of a similar nature are present between the muscle fibers in the abdominal wall and diaphragm. Large subcutaneous hemorrhagic areas are sometimes seen, especially over the shoulders and back to the loin. Epicardial hemorrhages are present and the pericardium may contain some straw-colored fluid. The lymph glands are usually not affected; however, a few may be swollen and red. There is usually inflammation of the abomasum and small intestine. The presence of whole grain in the fourth stomach is of considerable diagnostic value.

Care should be taken to differentiate between the large, blotch-like hemorrhages of this condition and the smaller, petechial hemorrhages of hemorrhagic septicemia.

Diagnosis.—When strong, fat lambs are dying on full feed and no evidence of bacterial diseases is present, this condition can be diagnosed. Withholding the grain ration will always stop the loss. Emphasis is placed upon the fact that overeating is frequently confused with hemorrhagic septicemia and treated as such by the administration of bacterins. Whatever benefits are gained from such treatment can be attributed to the reduction of feed during the time of treatment rather than to the effect of the bacterin itself.

Control.—It is important that concentrates be given only in amounts that can be tolerated. The physical condition of the lambs must be watched at all times for signs of indigestion and scouring, which are signals of approaching trouble. The margin between the fattening ration and the killing ration is slight and lambs once "sensitized" can be placed on full feed only with difficulty.

The lambs should be sorted so that each pen contains animals of a uniform size. If the animals are fed in troughs, not more than 12 inches should be allowed to a lamb per side. Even distribution of the grain in the trough is essential, in order that no lamb will get more than its share. The practice of lambing down corn fields and pea fields has been entirely abandoned in Colorado because our inability to control the amount of grain consumed resulted in excessive death losses.

Treatment.—Individual treatment is of little value and seldom possible because death ensues quickly.

INDIGESTION AND DIARRHEA

Lambs are extremely sensitive to indigestion and it may be observed in some form at any time during the feeding period. The sudden change from grass on the range to the hay and grain ration of the feed pen produces many cases of indigestion during the early feeding period. Later, as the grain ration is increased, diarrhea is seen even before the condition of overeating is recognized.

Too much feed, and frozen, mouldy or otherwise damaged feed, will cause indigestion and diarrhea.

Laxative doses of Epsom salts or mineral oil followed by hospitalization, with special attention toward the removal of the cause, constitutes the treatment.

PARASITES

External parasites are no longer of much importance in feed-lot diseases. Government regulations have so nearly stamped out scab that it is seldom seen in the feed lots.

The sheep tick, which is not a true tick but, rather, a degenerate fly, is quite common and if present in large numbers, causes intense irritation which prevents a normal gain. When present in sufficient numbers, they can be destroyed by dipping the animals twice, in coal-tar creosote or nicotine dip, at 28-day intervals.

Internal parasites are seldom the cause of death losses in the feed lot. Stomach worms and the broad tapeworm (*Moniezia expansa*) do little, if any, damage. The fringed tapeworm (*Thysanosoma actinioides*) is the most common, some flocks showing almost 100 per cent infestation. These worms are found in the small intestine, near the point where the hepatic duct enters; often, they ascend into the ducts of the liver itself. In recent years, many feeders have attributed the loss from

overeating to the presence of these worms. The administration of vermifuges have not only failed to remove the worms but in many instances caused severe death losses in the flocks.

The real loss suffered by the sheep-feeding industry from the increasing incidence of these worms is the economic value of the livers destroyed in the packing houses. This loss is estimated at a quarter of a million dollars annually, with nearly 30 per cent of all sheep livers condemned. Until more knowledge concerning the life cycle of this tapeworm is gained, nothing can be done to prevent its occurrence in feeder lambs.

Exogenous Hormones

A noted authority on metabolism in domestic animals (Simonnet) has remarked that vitamins are "exogenous hormones." The vegetable kingdom furnishes vitamins and the animal kingdom hormones, and the function of both of these groups is to stimulate, inhibit or regulate biological processes, some in a specific and others in a general rôle. The coöperation of vitamin E and the genital hormones has fathered the thought.

Arvey and Kokas have demonstrated that wheat germ oil produces hypertrophy of the uterus in virgins but not in spayed females used as checks. This appears to prove that vitamin E acts through the intermediary of the endocrine glands, perhaps the anterior pituitary, which has such a remarkable control over the process of reproduction. That wheat germ oil is gonadotropic and estrogenic has been apparent in clinical work but the mechanism of its action was never clear until this direct action on the body of the uterus of virgin females was demonstrated. It was thought to act only by nervo-muscular stimulation until this relation to the endocrine chain was established.

The "exogenous hormone," whether a hormone *per se* or a producer of hormones, would be welcome in clinical veterinary medicine, where such products, extracted from the organs of slaughtered animals, are too costly for general use. It is cheaper, for example, to extract vitamin E from wheat germ than to produce dependable extracts of the pituitary or other endocrine glands.

Anaphylaxis

That anaphylaxis is due to the presence of a substance in the blood of the sensitized animal is proved by the ability to sensitize a nonsensitized animal by transfusing blood from the former to the latter. Rickert named the substance toxogenine. The toxic substance produced by the action of the toxogenine of the blood upon the sensitizing protein, he called apotoxine, or anaphylactic poison.

The anaphylactic phenomenon can be demonstrated *in vitro*, for when toxogenine-sensitized blood is mixed with the sensitizing protein, the resulting mixture injected into a nonsensitized animal produces an anaphylactic reaction immediately.

This theory of anaphylaxis, which is quite generally admitted at the present time, was eclipsed for many years by the physical theory of Widal and others, who regarded anaphylaxis as a breaking up of the colloidal equilibrium of the blood and cellular protoplasm.—*Excerpt from Recueil de Médecine Vétérinaire, cxv, September 1939, p. 516.*

Nicotinic Acid in Delirium Tremens

From Mainzer and Krause (*Brit. Med. Jour.*, Aug. 2, 1939) comes the interesting information that nicotinic acid cuts short the duration of attacks of delirium tremens. A dose of 0.6 Gm. (9 gr.) made the symptoms of a severe attack disappear in twelve hours, after large doses of thiamin chloride had been given without effect.

Sixty cents a quart for milk, $2 a dozen for eggs, and $3 a pound for steak are the prices one would pay for these necessities of life if the wages of those producing them were equal to that of workmen in the building trades.

Brucella Melitensis Infection in the Maltese Goat*

By JOHN B. POLDING, B.Sc., M.R.C.V.S.
Valletta, Ghammieri, Malta

FOR MANY years the Maltese goat has been recognized as an animal which may be infected by the virulent strains of *Brucella melitensis* found in Malta. Nevertheless, it has been claimed that this host is sufficiently resistant to systemic infection to be classed as a "carrier" only and it has been denied that true clinical infection exists. Indeed, herd owners claim that rather than symptoms of disease arising, the infected animals are instead the most productive in the herd. Additionally, scientific workers state that abortion does not occur and that a bacteremia does not arise.

The reasons for this outlook are easily explained. First, there is no up-to-date work reported on this disease in the Maltese goat and, second, the herd owners in Malta are reluctant to part with any information about their animals. In fact, the only significant statement that they make is that goats found to be infected are usually the best milk producers in their flocks. Their reason for saying this becomes clear when it is explained that the diseased goats are slaughtered and compensation, depending upon the value of the animal, is allowed.

Preliminary work of the Undulant Fever Research Station, recently established in Malta, has altered the concept of this disease and it is gradually becoming obvious, in experimental animals at least, that as in the case of virulent local infections in man, the disease in the goat is probably the most consistent and acute of all the Brucella infections of animals.

The ensuing paragraphs survey the disease as it has appeared in experimental conditions through two breeding seasons in Malta.

*From the Undulant Fever Research Station, Valletta, Ghammieri.

The Mature Goat

RECOGNITION OF BR. MELITENSIS INVASION IN THE MALTESE GOAT

The mature pregnant goat, artificially infected by the subcutaneous injection of a smooth strain of *Br. melitensis*, offers the first measurable response to invasion by the change of the blood-serum-agglutination reaction. The change is preceded by a lag period during which there is no reponse and which, on the average, lasts about 72 hours. Thus, a low titre may be expected on the third day after invasion. From this point serum changes are rapid, the titre rising within the next 48 hours to a dilution of about 1:640. Thereafter, the rate of change slows and peak titres of 1:2,560 are reached by the eighth to the twelfth day. In our observations, 16 goats infected in this manner showed little individual variation.

In pregnant goats exposed to infection by contact, evidence of invasion in about 95 per cent of the animals is again offered by serum response. A small error occurs, as a few such animals, failing to agglutinate, on rare occasions prove to be slightly infected by subsequent isolation of small numbers of the organism. Out of 32 contact goats, two failed to offer a serum reaction when tested weekly, yet *Br. melitensis* was, on one occasion each, isolated from the milk. The first serum change among the contact animals was seen within the first fortnight of exposure, although this change has been delayed for as long as 100 days. The majority of first responses, however, are seen toward the end of the first month of exposure. The serum response in these animals, unlike that in those artificially infected, takes the form of a slow, irregularly rising titre with preliminary subsidiary peaks. In most cases final peaks are at approxi-

mately 1:2,560 dilution, and the rise to the final major peak usually coincides with the termination of pregnancy.

If the exposed animals are not pregnant, the response takes the form of a low (1:20-1:80) rise in serum titre. These responses are usually transient, a serum being 1:20 one week and negative thereafter. Occasionally, however, cases may occur where responses are moderately high (1:160) and the subsequent decline lasts for some months. It is thought that animals exhibiting such a response may have been in the early stages of pregnancy and aborted unseen.

Among contact-infected animals the long periods that sometimes elapse between exposure and first agglutination response do not necessarily represent the actual period between invasion and first serum change. From a comparison with the rapid response of artificially infected goats, and from the fact that the response of some of the contact-infected animals occurred as early as nine days, one is led to the conclusion that the first evidence of successful invasion into contact animals might be expected within a fortnight following satisfactory bacterial ingress.

PERIOD OF INCUBATION OF BRUCELLOSIS IN THE MALTESE GOAT

Reverting to artificially infected pregnant adults, it has been said that the serum response reaches a peak between the eighth and the twelfth day. At a point just before this peak, a bacteremia commences and this persists for more than a month. We have observed that when such animals are placed in contact with disease-free goats, transmission of the disease has at times occurred within nine days of artificial infection. Allowing three days for response to the serum in the recipient, one must assume that these recipients became infected within six days following the artificial infection of the donors; conversely, artificially infected goats become infective within six days following inoculation. Defining the period of incubation as the time that elapses between invasion and generalized systemic infection, with possible elimination of the organism, we may state that the period of incubation in artificially infected animals is between five and eight days.

The period of incubation in less heavily invaded contact-infected pregnant goats is more difficult to determine. The serum response does not always ascend with the same uninterrupted speed. Minor peaks form before the titre reaches its limit, although the final height of the curve is usually about the same as that of the artificially infected goats. Bacteremia is slight and intermittent at first. Longer periods elapse before generalized infection has become thoroughly established, and infection of the genital secretions supervenes.

In nonpregnant animals, it is doubtful whether true generalized infection occurs. If it does, it is probably short lived. In these goats serum response is hesitant and low, rarely lasting more than six weeks, when a return to negative or almost negative is the rule. It is believed that puerperal activity is essential for the full development of the disease.

Reverting momentarily to artificially infected goats: It has been noticed that such animals have transmitted the disease after injection but prior to signs of abortion. It must be assumed, therefore, that their genital secretions become infective before pregnancy terminates. This is an important point to the field worker.

CONFIRMATION OF BRUCELLA INFECTION IN THE MALTESE GOAT

In animals whose serum response is high, confirmation of infection presents no difficulties. Such animals are almost invariably pregnant, or have recently kidded. It has already been noted that it is probable that vaginal secretions are infective prior to parturition. Vaginal swabs during this period would probably, on culture, reveal *Br. melitensis.* In twelve artificially infected goats and two goats infected by contact, blood cultures proved the existence of a bacteremia from the time of the first serum peak. It may be noted that the bacteremia persists for a considerable

period, and confirmation may be effected at this time. At the termination of pregnancy *Br. melitensis* can be isolated regularly from the placenta and fetus of this class of goats, and thereafter the milk and the vaginal secretions carry the organism for weeks.

In pregnant or recently kidded animals whose serum response is slight or irregular, it may be necessary to resort to tissue culture and, in the case of milk and vaginal secretions, cultures may be required every day, from the first opportunity after kidding, before infection can be confirmed. Despite frequent cultures, in two out of 23 cases the organism was not recovered in goats of this description under experiment at this station, although serum response was present.

Confirmation in nonpregnant goats has not as yet been attempted.

CLINICAL SYMPTOMS

In Brucella infections the most compelling clinical symptom is abortion. Although the economic importance of abortion can not be argued, it seems that the abortion itself has received far too much attention from the epizoötiological standpoint. At any rate, the incidence of abortion in naturally infected animals seems largely fortuitous, and it is felt that a true insight into the nature of the disease can not be gained where abortions alone are counted and bacterial invasion is disregarded. Some authorities deny that abortion follows *Br. melitensis* infection in the Maltese goat.

In experiments carried on at this station, of 15 pregnant artificially infected goats, all aborted; of 22 pregnant females exposed to invasion by contact, nine aborted. Four of these animals were but slightly infected, having low serum reactions and eliminating the organism in smaller numbers and for a shorter period than many animals that had kidded at full term. On the other hand, six goats acutely infected, with high serum reaction and frequent heavy elimination of *Br. melitensis*, kidded normally. It is quite probable that there is some factor that controls the occurrence of abortion, but until it is completely understood, it seems useless to rely on abortion as an index of the extent of infection.

Clinical symptoms other than abortion exist in all acutely infected goats. Pyrexia occurs within 48 hours after generalized infection sets in. The animals lose condition rapidly and stand with a dejected expression, drooping head and staring coat. Pregnant goats become uneasy and, from their constant biting at a region high in the left flank, appear to be in pain at this point. There may be a slight diarrhea. In lactating goats the milk loses its normal characteristics and becomes a clear serum with suspended clots. While this milk degeneration remains, cultures of the milk serum yield a confluent growth of *Br. melitensis* in pure culture. Retention of the placenta has not been observed but a copious discharge from the vagina is common for two to three weeks after kidding.

These symptoms are most noticeable when infection and kidding occur early in the year—during the cold season of January, February and March. Animals kidding and becoming infected as late as June are less gravely affected and recover more rapidly. Hence, the peak individual rate of elimination of *Br. melitensis* may be placed early in February, and since this is the time when most goats kid, it is also the time when the greatest absolute elimination occurs.

It may seem odd, therefore, that the peak of human infection is reached during the months of July and August, but this can be explained in the following way. Goats kidding in February are generally not used for commercial purposes for about two weeks, owing to the presence of colostrum and the suckling of the kid. The greatest invasion to man will thus occur about March. It has been said that a three-week incubation period, at a minimum, is required in man. Among the Maltese (who possess a fair degree of racial immunity) the period may be much longer. Theoretically, early symptoms may be expected at the end of March or the beginning of April, but in actual fact these are not noticed until the inception of hot

weather—at the end of May or the beginning of June. Finally, public health records, from which the peak period in man is determined, do not show when the disease was contracted. Such records show only when the malady was reported to the health authorities, that is, when the patient entered the hospital or visited a doctor. The Maltese usually hesitates to seek medical aid until he becomes seriously ill, and since the onset of the disease is probably insidious in the racially immune, attention is not called to the condition until July or August.

PERSISTENCE OF BRUCELLA INFECTION

Although the blood-serum response may be taken as an indication of invasion by Brucella organisms, it is difficult to determine exactly when the infection has terminated. It is true that infection is no longer of interest to the epizoötiologist when the organism ceases to be eliminated from the body, but there is no way whatsoever of determining accurately whether such cessation has a sufficiently definite relationship to the fall or disappearance of serum agglutinins to serve as an authoritative guide. Indeed, once goats have suffered from an acute, generalized infection, they seldom regain an absolutely negative blood serum during the remainder of their useful life.

The peak serum reaction of an acutely infected animal persists, with minor variations and a slow, overall decline, for at least the period of lactation following infection. For example, goats becoming infected in February of 1938 and responding with a serum peak titre of about 1:2,560 reached, on the average, a serum reaction of 1:640 in August of the same year. Many even retained a titre of 1:2,560 in that month. Goats infected in the early months of 1937, having completed a further pregnancy in 1938, still retained titres of approximately 1:160 in the summer of 1938. Animals infected during 1929-1931 had titres of about 1:80 as late as 1936. Ten such animals were slaughtered and the tissues cultured. *Br. melitensis* was isolated from the supramammary gland of one animal.

Animals less severely infected ordinarily show the same proportionally slow decline over similar periods, but since their original peak titres would be in the region of 1:160, at the end of 18 months the serum would react in the rather neglected orders of 1:20 to 1:80. A small number of moderate reactors, however, and almost all nonpregnant reactors return to a negative serum reaction after a lapse of two or three months. This may represent recovery. One is inclined to doubt, on the other hand, that animals with a small or recurrent serum titre have made a complete recovery, and it seems reasonable to suspect that a persistent serum reaction following generalized infection represents a latent focus of infection, although this may be difficult to demonstrate.

In an attempt to throw further light on this problem, efforts have been made to determine the period during which *Br. melitensis* is eliminated in the secretions of goats that have become systemically infected while pregnant. It must be remembered that even in the most acutely affected goat, when the height of infection is passed (usually within a month), elimination becomes sporadic. For this reason, single negative cultures are worthless, and even daily cultures of the milk and vaginal secretions over long periods must be regarded as only moderately informative. In a group of twelve goats, the milk was plated every third day during the first, third and fifth month of the lactation following the pregnancy during which they became infected. All but one returned positive plates on the majority of days of the first month. There was some falling off in the rate of elimination toward the end of the month but the number of colonies derived from approximately 0.2 cc. of milk was generally more than 100. The milk had degenerated in character in all but three animals. During the third month only five of the goats returned colonies—two once, one twice, on on three occasions and one on five occasions. Colonies as a rule numbered less than ten. During the fifth

month, three of the five goats eliminating during the third month showed less than ten colonies on two or three occasions.

From these findings it will be seen that elimination can be regarded as constant only during the first four or six weeks of lactation. Thereafter, most goats apparently cease to produce the organism, while the few that continue to do so offer few colonies, and then but on rare occasions.

A second group of ten goats was similarly examined during the lactation following the second infected pregnancy. Of these, one remained a fairly constant eliminator of *Br. melitensis* up to the fifth month of lactation. Two others each returned less than ten colonies on one occasion during the first week of that lactation but at no time thereafter. The organism was not recovered from the other seven goats. During the first lactation of infection all of the ten had been constant eliminators.

The vaginal secretions of the two groups were cultured in parallel with the milk. The results were similar, but elimination in these secretions ceases earlier, since no positive cultures were recorded during the fifth month after termination of pregnancy. The genital secretions and the placenta of goats terminating their second pregnancy of infection do not usually appear to return the organism in culture, although cases of this have been observed.

Bearing in mind the bacteremia already mentioned, we may say, in conclusion, that acute, generalized melitensis infection of mature Maltese goats commences to localize during the second month after the termination of the pregnancy during which they were infected. In the majority of cases localization appears to exclude the udder and uterus by the fifth month after the termination of pregnancy, but a minority of goats retain genital infection to the end of this lactation. As a rule, the termination of a second pregnancy after infection will not cause an exacerbation of the disease but a small proportion of the animals will carry over elimination of *Br. melitensis* in the genital secretions to the second lactation. Considering all available data, it is the writer's opinion that infection may remain localized in the glands draining the genital areas for several years following acute infection.

The Immature Goat

RECOGNITION OF INVASION AND INFECTION IN THE KID

Evidence of invasion by *Br. melitensis* in kids can usually be obtained from the blood-serum-agglutination reaction. Confirmation by culture, except in the cases of the aborted fetus or still-born kid, is not practicable without slaughter, unless an exclusive study of the kid permits frequent blood culture at the peak of the serum reaction, or specialized methods of seeking *Br. melitensis* in the urine or feces.

An attempt has been made, however, to find the relationship between the cultural confirmation of infection in the fetus and the degree of infection in the dam. In such cases cultural confirmation has been readily effected when abortion has occurred in an acutely or moderately infected dam. On the other hand, where abortion occurred in two slightly infected dams, the fetus did not prove to be infected. When kids are carried to full term, and are still-born or die shortly after birth, confirmation has been effected only when the dams were acutely infected. Infection in the fetus, therefore, seems to be related to the degree of infection in the dam rather than to the occurrence of abortion. The results of culture of fetal membranes appear to run parallel to fetus infection. In our experience, at any rate, infection did not appear with greater frequency in the placenta than in the fetus. It thus appears that when infection is acute, the fetus and membranes will become infected and premature birth will commonly follow. It may happen, however, that an acutely infected dam is carried to full term, in which case it is infected when born. These offspring are either still-born or are weakly and die.

Kids of infected dams that appear to have escaped prenatal invasion and survive have been examined for the presence

of serum response only. In these cases few have been bled at birth prior to suckling, owing to the difficulties of obtaining blood at this time.

Nearly all kids have been tested, first between three and four weeks of age and, later, at monthly intervals. From these tests it is concluded that, with very few exceptions, surviving kids of infected dams are negative when tested at approximately 3 weeks of age despite the fact that they were suckling milk containing high whey agglutinins and enormous numbers of melitensis organisms. Additionally, these kids were exposed to the same kind of contact infection that proves capable of infecting 85 per cent of adult animals around them. Kids, therefore, seem highly resistant to infection for about two months following birth. Also, they do not, as is suggested by some observers, appear to derive agglutinins from the maternal milk. After two or three months of life the kids become less resistant and serum response develops in some.

Persistence of Infection in the Kid

Kids that develop a serum reaction through natural infection either before birth or during the third or fourth month of life seldom retain blood agglutinins for long periods, and the response curve is similar to that of naturally infected nonpregnant adults. Rapid recovery is the rule and clinical symptoms are not usually noticeable. This is rather to be expected, as no question of puerperal activity can arise in animals of this age. Kids artificially infected during the fourth month of life, on the other hand, show some clinical symptoms of disease, in the form of retardation of growth and, sometimes, slight fever and diarrhea. Although such kids will show a rapid and high agglutination rise, the serum curve will fall off equally rapidly after reaching its peak and a negative serum will be returned by the time the kid is 8 months of age. This serum change is comparable to that of artificially infected nonpregnant adults, with the exception that the adult curve will not always return to negative and will fall off at a much slower rate. Kids artificially infected at 5 or 6 months of age will again show clinical symptoms and a rapid high serum rise. In their case the fall of the serum curve, like the artificially infected nonpregnant adult, is slow and the kids will return a positive serum in the region of 1:10-1:40 at 12 to 14 months of age.

In general, it may be stated that the older the kid, the more permanent will be the serum reaction, that this permanence will be proportional to the magnitude of the infecting dose, and that the critical age after which, when infection is great, the serum will not rapidly return to negative is about 5½ months.

Conclusion

Contrary to popular conception, the Maltese goat is not unique in its resistance to *Brucella melitensis* infection. The disease picture in this animal not only runs parallel to Brucella infections in other animals but also appears, under experimental conditions at least, to offer the most regular and vivid history of brucellosis yet reported. This statement seems justified in view of the facts that goats are often found so severely infected that the milk becomes degenerate, a state unusual in other animals, and that grave clinical symptoms, seldom reported in cattle, frequently arise.

Hydatid Disease in New Zealand

In 1938, there were reported 1,304 cases of hydatid disease with 151 deaths in a population of 1,604,244. It is stated that there are approximately 31 million sheep and 4½ million cattle in New Zealand and that the livers and lungs of more than one half which reach adult life are studded with cysts of Echinococcus or Tenicollis types —in many cases, both. The prevalence of the adult parasite (= *Taenia echinococcus*) in the dog is very high. Among the many features of the prevention campaign was the distribution to dog owners at the time of licensing of a supply of arecoline vermifuge tablets. (*Journal of the American Medical Association, cxiii, December 9, 1939, p. 2165.*)

A Histological Study of the Mammary Gland of the Cow During Pregnancy*

By FRANCIS J. KWONG, B.S., D.V.M.

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BIZZOZERO and Vassale[1] were the first observers of the histological changes of the mammary gland during pregnancy. They declared that there is an enormous multiplication of epithelial elements during pregnancy by mitotic division of cells. This observation has been confirmed by all who have done research on this subject. They also stated that the proliferation of the epithelial cells gives rise to numerous alveoli, of which the wall is formed by a single layer of epithelial elements.

Benda[2] was able to follow the formation of the epithelium during pregnancy in the cow. He found that in the resting stage of the mammary gland there is a layer of cells just outside of the alveolar epithelium continuing down along the ductules. During lactation this external layer disappears and is represented by a myoepithelial layer which takes up the active rôle as the mechanism of milk secretion.

Lacroix[3] corroborated Benda's statement by his findings in the mammary gland of women that there is such a cellular layer external to the alveolus during pregnancy. Kolossow[4] also made observations and interpretations of these elements. He further found that at the final stage of pregnancy, these elements transformed into typical muscle fibers. Winckler[5] described this layer as the membrana propria, or basement membrane, composed of a transparent membrane with elongated connective tissue cells. Kolesnikow[6] considered it a membrane made up of flattened, stellate, connective tissue cells united by means of their processes. Rauber[7] described it as an endothelial lining. According to Jakowski,[8] this membrane is formed by several layers of anastomosing cells.

Brouha,[9] from whose work the foregoing review of literature was taken, observed in several species the double-celled epithelial layer of the mammary gland before and during the early stage of pregnancy and stated that when the gland activity begins, a single layer only is visible. He also stated that the formation of the double-celled epithelial layer is due to the multiplication of cells and that, later, the cells of the external layer are stretched longer and longer, finally becoming discontinuous. He could not find any evidence to support Benda's[2] statement that these cells transformed into smooth muscle fibers.

Hammond[10] described the different stages of development of the cow's udder during pregnancy. He reported that in the first three months of pregnancy the development is, as a rule, limited to the duct system only and that this develops in and along the connective tissue bands which divide up the fat of the udder. At about the fifth month the character of the epithelium of the alveolar duct begins to change and those parts of the alveoli which have completed their growth in length now begin to develop in diameter. Then, glandular activity begins.

Cole[11] investigated the mammary gland of the mouse during pregnancy and Jeffers[12] studied the cytology of the mammary gland of the albino rat during pregnancy. Dawson[13] observed the histological structure of the mature gland of women in pregnancy. He stated that at about the third month of pregnancy, the newly formed ductules begin to throw off the superficial part of their two-lining cell layers and the basal or external layer remains as a unicellular lining.

MATERIAL AND TECHNIC

Hammond's[10] measurements were followed in estimating the stage of pregnancy.

The udders used in this study were: One from a heifer in the first month of

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pregnancy; one from a cow in the first month of pregnancy; one from a heifer in the third month of pregnancy; one from a cow in the fourth month of pregnancy; three from cows in the fifth month of pregnancy; one from a heifer in the sixth month of pregnancy; two from heifers in the seventh month of pregnancy; three from cows in the seventh month of pregnancy; two from cows in the eighth month of pregnancy; two from heifers in the ninth month of pregnancy; one from a cow in the ninth month of pregnancy; two from nonpregnant heifers; and two from nonpregnant, lactating cows.

One of these udders was collected from the meat laboratory of the College and the others were obtained from the Iowa Packing Company, Des Moines.

The paraffin method was employed. Specimens were prepared 7μ (.000275 in.) in thickness and stained with hematoxylin and eosin, iron hematoxylin, and Mallory's triple stain.

DESCRIPTIONS OF UDDERS

Udders of Nonpregnant Heifers for Comparison. — There is no secretory or alveolar tissue in this stage. The ductules are situated in groups amid the fat tissue and their epithelium has two layers of cells with a basement membrane. The internal or superficial layer is columnar in the type of cells, and the cells are closely packed. The external or deep layer is rounded, cuboidal, or flattened. The connective tissue is found mostly surrounding the ductules in groups, which is full of cellular elements and is composed of white fibers. Connective tissue bands separate the fat masses. These bands consist primarily of white fibers with comparatively less cellular elements. Fat tissue is abundant (fig. 1).

Udders of Pregnant Heifers. — a) 3-month-pregnant stage: The udder tissue of this stage is similar to that of nonpregnant heifers. No secretory tissue can be found. The ductules are at their height of proliferation.

At the first month of pregnancy the connective tissue bands are denser and more numerous in comparison with those of the nonpregnant heifer. The fat tissue seems to be decreasing in amount, being gradually replaced by white, fibrous tissue. The epithelium of the ductules still appears two-layered with a basement membrane (figs. 2 and 3).

At the third month the fundamental duct system has been well laid down. The secretory tissue has not yet been developed but the elementary lobes and lobules can be traced by the groupings of the ductules. The character of the epithelial cells of the ductules is the same as that mentioned in the preceding paragraph. (See figure 4.)

b) 4- to 7-month-pregnant stage: In this stage the secretory tissue is formed and begins to function. Three specimens were examined, one in the sixth month of pregnancy and two in the seventh month, but their development was not in accord with the age of pregnancy. One of the seven-month-pregnant specimens seemed retarded in its development and thus appeared younger than the other two. Individual or breed differences might have accounted for the variation. However, by reversing their ages these three specimens serve to illustrate fairly well the development in this stage.

In the early part of this stage the alveoli are in the active stage of formation by means of proliferating and expanding the end buds of the ductules. Both lobes and lobules are well differentiated. Most of the newly formed alveoli are in the form of a mass of cells with or without a small lumen. Their outline is not clear but it is detectable. There is loose connective tissue and cellular elements surround them. The number of layers of alveolar epithelial cells varies and the cells of the basal layer or layers are irregularly arranged. Therefore, the basement membrane is indistinct. Most of the ductules still have two layers of epithelial cells. The columnar cells of the internal layer in these ductules become shorter (fig. 5).

A minute amount of secretion may be seen in the lumens of some of the alveoli, ductules and large ducts (fig. 7). Fat cells are rare. The lobes are small, and the in-

terlobar connective tissue trabeculae are very wide. These give room for the fullest development of the alveoli.

In the later part of this stage the alveoli are full of secretion, which is a good indication of completed development. The epithelial cells are cuboidal and in a single layer. The basement membrane is a thin layer of connective tissue fibers with flattened cells imbedded in it. The ductules still have two layers of epithelial cells (fig. 6). The cells in the internal layer are apparently cuboidal in shape, while those of the external layer are flattened, polyhedral, or elliptical and, thus, irregularly arranged. The ductules also are full of secretion. The interalveolar spaces are filled with loose connective tissue fibers, cellular elements, vessels, lymph spaces and nerves. The interlobular spaces are filled primarily with narrow bands of dense connective tissue, and the interlobar spaces with wide bands of dense connective tissue.

c) 8- to 9-month-pregnant stage: The alveoli are large and can be compared with those of lactating udders. They are full of secretion, which is stained a striking red color with eosin. The alveolar epithelium and the basement membrane are the same as those described in the above paragraph. In most cases the interalveolar connective tissue is thin and is filled with a narrow strip of white fibers. The ductules are lined practically with one layer of epithelial cells. However, an external or basal layer of discontinuous cells may be found here and there. The interlobular and interlobar connective tissue are narrow, which indicates that the alveoli have been fully developed and distended by secretion (fig. 8).

Udders of Pregnant Cows.—Ten udders from pregnant cows at different stages of pregnancy were examined. They were all like lactating udders.

Udders of Nonpregnant, Lactating Cows for Comparison.—The udders of this stage are much like those of the final stage of pregnancy and those of pregnant cows (fig. 9).

DISCUSSION

In the first three months of pregnancy in the heifer, the udder tissue is similar to that of the nonpregnant heifer. It is the stage of proliferation of ducts and no alveolar tissue could be found. This agrees with Hammond's[10] and with Turner's[14, 15] findings. There are definitely two layers of epithelial cells of the ductules in nonpregnant heifers as well as in all stages before the ninth month of pregnancy. At the ninth month of pregnancy or, rather, the last one or two months of pregnancy, and also in the lactating period, the remnants, or the discontinuous elements of the external layer, were found in the writer's material (fig. 8). Therefore, this finding supports Brouha's[9] view. The stage of pregnancy at which the character of the epithelium of ductules begins to change is different from Hammond's[10] account.

There has been much controversy about the origin and nature of the perialveolar layer of cells, called myoepithelium or basket cells. Benda[2] thinks that this layer of cells represents the original external layer of the two-layered ductule. McCarty[16]

CAPTIONS TO ILLUSTRATIONS ON OPPOSITE PAGE (LEFT TO RIGHT, TOP TO BOTTOM)

Fig. 1. Udder of nonpregnant heifer, 2 years old (x 133). 1) Ducts; 2) fat cell; 3) connective tissue band.

Fig. 2. Udder of a one-month-pregnant heifer, 2 years old (x 133). 1) Ducts; 2, 3) two layers of duct epithelium; 4) dense connective tissue; 5) fat cells.

Fig. 3. A duct from the udder of a one-month-pregnant heifer, 2 years old (x 533). 1) Lumen of duct; 2, 3) two layers of duct epithelium.

Fig. 4. Udder of a three-month-pregnant heifer, 2 years old (x133). 1) Ducts; 2) fat cells.

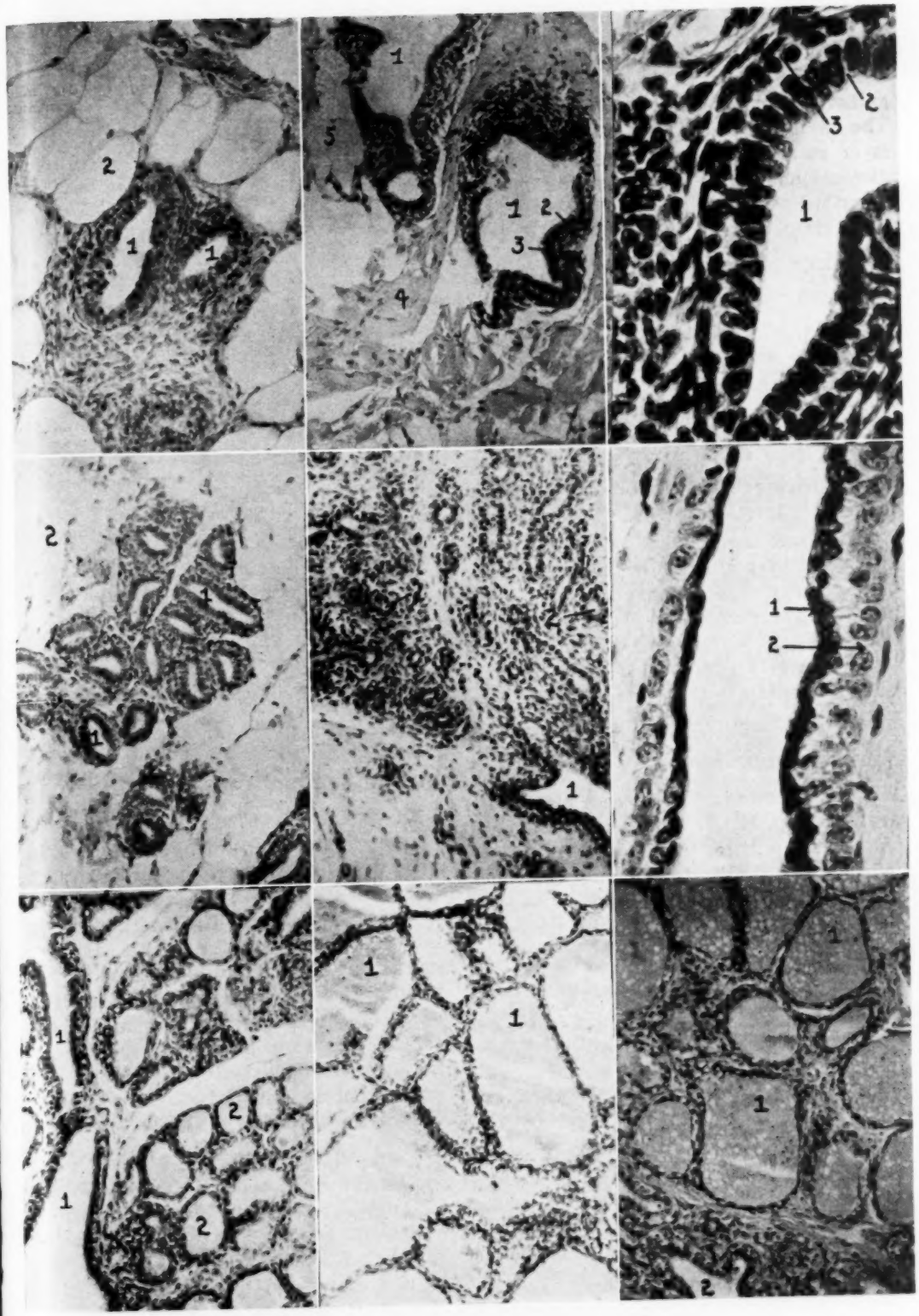
Fig. 5. Udder of a seven-month-pregnant heifer, 4 years old (x 133). 1) Duct; 2) alveoli in formative stage.

Fig. 6. A duct from the udder of a seven-month-pregnant heifer (x 533). 1, 2) Two layers of duct epithelium.

Fig. 7. Udder of a seven-month-pregnant heifer, 2 years old (x 133). 1) Duct; 2) alveoli.

Fig. 8. Udder of a one-month-pregnant heifer, 3 years old (x 133). 1) Alveoli much distended with secretion.

Fig. 9. Udder of a lactating cow, 3 years old (x 133). 1) Alveoli; 2) duct.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considered these cells as "generative" or "reserve" cells provided for regeneration. Kolesnikow[6] described them as connective tissue fibers with their stellate cells, together with the homogenous membrane. The writer prefers Benda's idea. This layer seems to be much the same as the discontinuous layer of cells of the ductules described above. They are all derived from the external layer of the original two-layered epithelium.

SUMMARY

The first three months of pregnancy comprise the period of duct proliferation. During the fourth to the seventh month of pregnancy the secretory or alveolar tissue forms. Secretion is observed in the later part of this period. In the last two months of pregnancy alveoli are much distended by secretion.

There are two layers of cells in the ductules during and before the early stages of pregnancy. At the later stages of pregnancy and during the lactating period the external layer is represented by the discontinuous layer of flattened cells in the ductule, continuing to the alveolus as the perialveolar layer of cells.

ACKNOWLEDGMENT

The writer desires to express his sincere gratitude to H. L. Foust, under whose direction this work was done. He is also indebted to C. W. Deming of the Iowa Packing Company, Des Moines, and to F. J. Beard for their help in collecting the material.

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Milk Inspection

In a small city a newspaper reporter recently attempted to learn the name of the milk dealer who was to be prosecuted because six successive milk samples showed a high bacteria count. The health officer told the reporter, "We never give out these names. It is unfair to the dealer, the competitor and to the public." Asked if the name of the dealer would be made public after the case was prosecuted, the officer replied, "If the magistrate wants to reveal the name, that is his affair."—*Harry Seal in Consumers' Digest.*

Vitamin D is concerned in reproduction, lactation, growth and development of the bones. Its sources are the sterol fractions of fats, of which cod liver oil ranks first. It was discovered by McCollum of Johns Hopkins University in 1922.

Methylene Blue as an Antidote for Poisoning by Oat Hay and Other Plants Containing Nitrates*

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A LARGE number of plants have been found to contain high concentrations of potassium nitrate due to conditions (with which we are not as yet acquainted) prevailing in the locality in which these plants are grown. When these plants are ingested by cattle, some of this nitrate is reduced to nitrite, which converts hemoglobin into methemoglobin. This methemoglobin is incapable of giving up its oxygen to the tissues and thus causes an anoxemia, the severity of which is proportional to the amount of hemoglobin converted.

The greatest loss in Wyoming due to plants containing high concentrations of nitrate occurs in oat hay and straw poisoning. [1, 2, 3, 4] One loss of three steers reported to us was traced to the animals' feeding on pigweed containing 3.6 per cent KNO_3. We have found high concentrations of nitrate in other weeds and in some specimens of sorghum grown in our experimental garden. South Dakota[5] reported deaths from sorghums which contained little or no hydrocyanic acid but which did contain 9 per cent of nitrates. Minnesota[6] reported several cases of sorghum poisoning caused by sorghums containing too little HCN to be toxic. Perhaps these sorghums, too, contained high concentrations of KNO_3.

Several investigators have reported on the use of methylene blue in combating the methemoglobinemia caused by sulfanilamide therapy. One of these investigators[7] showed that methemoglobin formed by the injection of nitrite into the blood stream of dogs rapidly disappears following the administration of methylene blue. For this reason, we decided to determine the efficacy of methylene blue in counteracting the methemoglobinemia produced in cattle by the ingestion of nitrates.

Thirteen experiments were made on ten head of cattle. These animals ranged in size from less than 91 kg. (200 lbs.) to more than 227 kg. (500 lbs.). Three animals drenched with varying doses of KNO_3 received no treatment and died. The doses given to these animals varied from 0.5 to 1.6 Gm. per kg. of body weight. Since as little as 0.55 Gm. of KNO_3 per kg. of body weight is a lethal dose, seven other animals were drenched with doses in excess of this quantity and, after going down, were treated with methylene blue.

The KNO_3 in all cases was dissolved in water and administered by stomach tube. After sufficient methemoglobin was formed to cause the animals to go down from weakness, blood was drawn and the methylene blue injected intravenously as a 4 per cent solution. At various intervals after the injection of the methylene blue, blood samples were taken. Methemoglobin analyses were made on all blood samples to determine the percentage of conversion of the hemoglobin to methemoglobin.

The data of these experiments are given in table I. By conversion to metHb is meant the percentage of the total pigment that is present as methemoglobin. Animals 1-3 received no treatment and died. The next three (4-6) received small quantities of methylene blue and had recurrences with symptoms typical of nitrate poisoning. Two of these (4 and 5) received no further treatment and died. Animal 6 received another injection of methylene blue and recovered.

All of the animals treated with methylene blue were back on their feet within ten to 20 minutes after treatment. Methemoglobin analyses made on the blood samples taken before and after treatment showed pronounced decreases in the amount of hemoglobin converted into methemo-

*From the Wyoming Agricultural Experiment Station.

TABLE I—Analyses of blood samples following injections of KNO_3 and methylene blue to determine the percentage of conversion of the hemoglobin to methemoglobin.

				Animal Down			After Injection of Methylene Blue	
Animal	Weight (Kg.)	Date	Amount KNO_3 Given (Gm.)	Conversion to MetHb (%)	Time (Hrs.)	Amount M. B. Given (Gm.)	Conversion to MetHb. (%)	Time (Min.)
1	126	3-20	141	80	7	0		
2	123	7-12	67	88	4.5	0		
3	91	9- 8	150	90	3.5	0		
4	91	9- 8	150	75	4	0.5	22	20
5	91	9- 8	150	77	3.5	0.6	43	15
							51	
6	227	9-13	300	80	3.5	0.6	51	15
							36	35
							46	120
							82	270
				82	8	2.0	42	20
							27	35
7	91	9- 7	100	80	9.5	2.0	0	15
6	227	8-28	150	73	5	2.0	13	40
8	100	8-28	100	74	8.25			
				78	9.5	1.5	13	20
							4	60
6	227	8-31	200	86	5.5	2.0	27	20
							4	60
6	227	9-11	200	79	8	2.0	0	15
9	250	9-19	350	80	6.5	2.0	40	15
							26	30
							7	70
10	205	9-19	300	77	7	2.0	33	15
							6	50

Results Following Administration of Methylene Blue.—Animals 4 and 5 recovered but symptoms recurred; died five hours later. Animal 6 recovered temporarily following an injection of 0.6 Gm. of methylene blue, but went down again; recovered following an injection of 2 Gm. Animals 7-10 recovered.

globin shortly after the methylene blue was injected. The values for methemoglobin after the injection of methylene blue are undoubtedly high, for there is sufficient methylene blue left in the blood to absorb light in the vicinity of the spectrum in which the determinations are made.

It would appear from the data that 2 Gm. of methylene blue is sufficient adequately to protect an animal weighing up to 250 kg. (550 lbs.) against the ingestion of 6.3 kg. (14 lbs.) of plant material containing about 5 per cent of KNO_3. Even though the data presented show no recurrence of symptoms in any animal treated with 2 Gm. of methylene blue, it would appear possible for this to happen, provided that the quantity of ingested nitrate is sufficiently great. Therefore, it is highly advisable to watch the treated animals for some time afterward. Of course, any forage found to contain high concentrations of nitrate should not be fed.

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Physiological Studies of Induced and Natural Bloat in Dairy Cattle*

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FOLLOWING the loss of four valuable animals in the Oregon State College dairy herd on irrigated ladino clover pasture during the summer of 1938, some investigational work was started on the physiological aspects of bloat. In a preliminary report made in 1937, Cole and Hart suggested that the character of the gases formed in the rumen is more important than the mechanical pressure factor. The effect of insufflation with the gases was tried, the various gases alone and in combinations being used.

It was deemed advisable, at first, to establish definitely the fact that gases are absorbed from the rumen and, then, to find the rate of absorption. Four sheep were used in this study. They were placed under nembutal anesthesia. The abdominal cavity was opened and citrated blood samples were taken, simultaneously, under mineral oil, from the portal and jugular veins. The volumes per cent of carbon dioxide was determined, the Van Slyke apparatus being used. Ligatures were placed around the anterior part of the abomasum near the omasal-abomasal orifice, thus confining the gas to the first three compartments of the stomach. The rumen was then insufflated with carbon dioxide. After allowing the gas to remain in the first three compartments for a period of one to two minutes, blood samples were again drawn simultaneously from the portal and jugular veins and the volumes per cent of carbon dioxide determined. The carbon dioxide volumes per cent increase ranged from 0.9 to 9.4 per cent. There was no appreciable change in the carbon dioxide content of the blood drawn from the jugular vein. Respiration increased in all cases during the carbon dioxide insufflation.

In order to determine in what manner mechanical pressure alone affected the animal, a cow with a rumen fistula was used, a special pressure plug being placed in the permanent fistula. Air was used in establishing various pressures ranging from 45 mm. to 90 mm. (1.75 to 3.5 in.) of mercury above atmospheric pressure. Since the animal eructated the air quite readily, a large quantity of water was pumped into the rumen and the posterior end of the animal elevated so that the water level was above the cardia. This prevented belching, but the animal, although showing some uneasiness, was in no distress at any time. Breathing, however, became quite shallow. Later, attempts were made to prevent belching by putting balloons against the cardia from a cord passed down through a stomach tube, but this method was ineffective. During the course of the gas-insufflation experiments, kymograph tracings were made of respiration and rumenal contractions.

Since an increase in pressure seemed to have no extreme effects on the animal, the various gases were used at first separately and then in various combinations. Carbon dioxide was used and it produced rather striking reactions, chiefly upon the respiratory apparatus, causing extreme dyspnea. Pressures were established as high as 90 mm. (3.5 in.), the symptomatology being confined to the respiratory apparatus. There was no rise in the volumes per cent of carbon dioxide in the jugular vein of the cow during carbon dioxide insufflation. The respiratory symptoms appeared from one to two minutes after the insufflation was started. Cheyne-Stokes type of respiration occurred in both animals.

Methane insufflation produced mild symptoms similar to those produced by air pressure. Symptoms induced by hydrogen

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insufflation were similar to those of methane and air pressure. Carbon dioxide and methane (40 per cent methane to 60 per cent carbon dioxide) produced symptoms similar to those of carbon dioxide insufflation, although not as marked.

None of these gases produced an appreciable change in rumen contractions except during the height of the pressure. The animal eructated quite readily, and as soon as the rumenal pressure had subsided, rumenal contractions increased in rate. The insufflation experiments were repeated on two cows at various intervals. Several sheep were used and experimental work was done to determine the effect of rumenal pressure on the heart rate and blood pressure. Direct blood-pressure recordings were made from the carotid artery of sheep under nembutal. Increased rumenal pressure caused a rise in blood pressure and an increase in heart rate, but even tremendous pressures had no other effects that could be detected by the methods used.

In an attempt to get more information about the rumen gases, analyses were made at various intervals from the cows with fistulas. After a series of analyses had been made, one cow was fed freshly cut ladino clover for a period of five days, following which another rumen-gas analysis was made. Carbon monoxide appeared in this analysis in an appreciable amount. Carbon monoxide determinations were made with the Orsot gas-analysis procedure, the slow combustion method being used. They were checked with the silver-nitrate-reduction test.

Duplicates were run on all gas analyses. This suggested further insufflation experiments, with a small percentage of carbon monoxide in the insufflating gas. At first, exhaust gas was used, establishing a pressure of 50 mm. (1.9 in.) sustained for five minutes. A gas analysis was made after the cow became prostrated and the carbon monoxide content of the gas obtained at this time was 7.4 per cent. The carbon monoxide content of the blood was 80 to 90 per cent saturated. The cow lived, but was in a semicomatose state for one-half hour after the plug was removed.

The blood was checked 24 hours later and found to contain no carbon monoxide hemoglobin. This work was repeated on another cow. In this case, the exhaust gas was pumped in slowly for three minutes. The intrarumenal pressure was built up to 50 mm. with carbon dioxide and maintained for seven minutes. This was repeated again and maintained for five minutes. After the last insufflation, carbon monoxide was found in the rumen gas in a concentration of 0.15 per cent. The blood at this time was 35 per cent saturated. The animal showed marked symptoms, which indicated that it was near the prostration point. The experiment was repeated. Exhaust gas was

Carbon dioxide content of sheep blood.*

Treatment of Sheep	Content CO_2			
	October			
	15	18	19	26
Blood from portal vein before CO_2 treatment	52.8	46.6	66.4	62.6
Blood from jugular vein before CO_2 treatment	51.4	42.8	63.6	63.6
Blood from portal vein after introduction of CO_2 under pressure into rumen	60.2	56.0	67.3	71.1
Blood from jugular vein after introduction of CO_2 under pressure into rumen	50.4	46.6	63.6	63.6
Blood from portal vein after introduction of CO_2 under pressure into intestine		64.5		

*The author wishes to express his thanks to L. D. Wright of the Oregon Agricultural Experiment Station for his work in making the CO_2 determinations.

CO_2 content of blood expressed as cubic centimeters of CO_2 at 0° C. and 760 mm. pressure per 100 cc. of blood.

pumped in for 2½ minutes and the intrarumenal pressure was built up with methane to 50 mm. Rumen gas showed a concentration of 1.2 per cent carbon monoxide, and the blood was 45 per cent saturated. Symptoms were noted in three to five minutes.

A small amount of exhaust gas was forced through the fistula of the cow, thereby raising the intrarumenal pressure to 50 mm. A combination of carbon dioxide and methane (60 per cent carbon dioxide and 40 per cent methane) was used. Marked preprostration symptoms appeared in about two minutes. The blood was 50 per cent saturated with carbon monoxide and the rumen gas contained 2.2 per cent carbon monoxide. As in previous carbon monoxide experiments, the rumen practically ceased all movements. Eructation was inhibited. Blood samples were taken for histamine determination at the same time that the carbon monoxide blood sample was taken. The test was negative.

DISCUSSION

Although it seems unlikely that in many cases the intrarumenal pressures that exist in acute bloat should be responsible for the death of the animals, the mechanical effect of pressure can not be ignored.

As intrarumenal pressure increases, the rate of gas absorption increases. If death in acute bloat is then due to the toxic effect of certain rumen gases, the rate of absorption of these gases depends somewhat on

Experiments on CO in rumen of cow.

DATE 1939	COW	TREATMENT	CO_2 IN RUMEN %	CO IN RUMEN %	CO IN BLOOD %	REMARKS
June 9	Jersey	Exhaust gas to 50 mm. Maintained 5 minutes ±		7.4	80–90	Choke on car out full; cow in coma
12	Jersey	Checked CO in blood			0	
13	Holstein	Poisoned with exhaust gas the previous afternoon; blood sample taken			0	
14	Holstein	Normal feeding; hay		0.2		
15	Jersey	Fed ladino clover overnight		0.3		
16	Jersey	Fed ladino clover 38–40 hours		0.3		
16	Holstein	Fed one day on clover		0.2		
17	Holstein	Fed two days on clover		0.2		
17	Jersey	On clover		0.3		
19	Holstein	On clover		0.0		Not feeding well
20	Holstein	On clover		0.3		Feeding satisfactorily
21	Holstein	Exhaust gas for 3 min. No pressure. Pressure built up to 50 mm. with CO_2. Maintained 7 min. Repeated. Maintained 5 min.		0.15	35	First symptoms of poisoning noted; choke on car in
23	Holstein	Exhaust gas for 2½ min. No pressure. Pressure built up with methane to 50 mm.		1.2	45	Symptoms noted in 3 to 5 min.; choke on car in
July 14	Jersey	3 to 4 min. exhaust. Pressure up to 50 mm. (60% CO_2 plus 40% methane)	31	2.4	50	Symptoms noted in 3 min.; cow near prostration

the intrarumenal pressure, which may be increased by the rapid formation of relatively harmless gases, such as methane, hydrogen and even carbon dioxide.

Little is known at present about bacteria that might produce carbon monoxide, at least under conditions that exist in the rumen.

The symptoms induced in this work by carbon monoxide insufflation supplemented with a rise in intrarumenal pressure with other gases are remarkably similar to the symptoms observed in actual bloat cases. The carbon-monoxide-insufflated animals and the actual acute bloat cases do not show the marked dyspnea noted in carbon-dioxide-insufflated animals. Carbon monoxide was the only gas used in this work that seemed to inhibit belching.

Although the intrarumenal pressures were not measured in the acute bloat cases observed, the pressures did not seem to be nearly as great as some of the induced cases (as high as 1.7 lb. above atmospheric pressure per square inch), and yet the animals died suddenly.

CONCLUSIONS

Gas absorption from the rumen is quite rapid; the greater the amount of rumenal mucosa exposed, the faster the rate of absorption.

Carbon dioxide is absorbed from the rumen and produces marked symptoms upon the respiratory apparatus, relatively high pressures causing extreme dyspnea. An increase in intrarumenal pressure causes an increase in the rate of absorption of carbon dioxide and carbon monoxide from the rumen.

Carbon monoxide, when present in the rumen in very low concentrations, will produce marked symptoms when the intrarumenal pressure is increased with other gases common to the rumen. The intrarumenal pressure in this case need not be extreme.

Carbon monoxide was found in an appreciable amount in two experimental cows fed freshly cut ladino clover.

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Tularemia Increasing

Signalized for the first time in 1910 by McCoy of the United States Public Health Service, tularemia is now rated as a "dread disease" in secular announcements, and in scientific circles its potentiality is not discounted. The disease is known to exist in all but two states—Vermont and Connecticut. During 1938, there were 2,081 known cases in the United States, which is four times more than the figures for any previous year. For the period 1924-1938, public health statistics show: Illinois, 1,510 cases; Ohio, 939 cases; Virginia, 793 cases; Missouri, 725 cases; and Kentucky, 690 cases.

The total number of cases for the United States in 1937 was 960. The tularemia situation in Iowa is an example of the way the disease is spreading. With but 74 cases reported for the period 1924-1937, the number for December 1938 alone was 82.

While tularemia is spread by other rodents, 90 per cent of all human cases are traceable to three breeds of wild rabbits: The cottontail, which inhabits all parts of the United States; the jack rabbit of the range country and Pacific coast; and the snow-shoe hare of Canada, California and the Southwest.

Though the discovery of *Clostridium tetani* is generally credited to Nicoliar, Rosenbach and Kitasato, it was Robert Koch who definitely identified the earth bacillus of these authors with the disease. The date of the discovery by Koch is given as April 10, 1887.

Correlation of the Brom-Thymol-Blue Test with the Bacteriological Findings in the Diagnosis of Mastitis*

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Newark, Del.

THE BROM-THYMOL-BLUE test has been recommended widely as a field test for the diagnosis of chronic mastitis. From a disease-control standpoint, the test was considered to be of value in detecting subclinical cases of mastitis where laboratory facilities are not available.

Because of the extensive use of this test as an aid in diagnosis, in judging the quality of milk, and in the control or eradication of mastitis in infected herds, a study was made in which the color reaction of the milk containing brom-thymol-blue was compared with the bacteriological findings. In correlating these results, the green-yellow or yellow-green brom-thymol-blue reaction is designated as normal hydrogen-ion concentration, while milk giving light blue, blue, dark blue, green, dark green and yellow indicates abnormal or mastitis milk.

METHODS OF STUDY

Milk samples were collected from individual quarters at weekly intervals from 48 milking cows, all of which were cases of clinical or subclinical mastitis in one or more quarters of the udder. The animals were members of three herds, two of which were purebred Guernsey and the third purebred Holstein-Friesian. The brom-thymol-blue reaction was compared with the bacteriological findings of 1,060 individual quarters.

The milk samples from the individual quarters were collected in sterile bottles and brought under ice to the laboratory, where they were cultured immediately on blood agar and tested with brom-thymol-blue. In collecting the samples the udders of the cows were washed with chlorine water, the teats were washed with 70 per cent alcohol, and the first streams of milk were rejected. There was only a short interval between the collection of the samples in the field and their testing at the laboratory.

The brom-thymol-blue test was made by adding 0.25 cc. (4 minims) of the stock solution of brom-thymol-blue to 2 cc. (0.07 oz.) of the milk sample contained in a clean test tube. The tube was then shaken and the resulting color immediately recorded. The stock solution of brom-thymol-blue was prepared by dissolving 100 mg. (1.54 gr.) of the dye in 3.2 cc. (0.12 oz.) N/20 NaOH and diluting to 250 cc. (8.3 oz.) with distilled water.

The cultures were made by streaking the milk upon blood-agar plates. The basic medium used in the plates was tryptose agar and citrate ox blood in the ratio of 10 cc. (0.33 oz.) of tryptose agar to 1 cc. (15 minims) of blood. The plates were poured in advance of the time when streaked. They were incubated for 24 hours to test their sterility and then stored in the refrigerator until used.

After having been streaked, the plates were incubated at 37° C. for 48 hours. After examination of the growth upon the original plates, suspicious colonies were fished and transferred to tryptose broth. These cultures were tested for purity, following which the physiological, cultural, and antigenic properties of the organism were studied.

RESULTS

The results of the tests were recorded as positive or negative. A positive brom-thymol-blue test was one wherein the milk assumed a color other than the yellow green, either in the direction of the blue- or yellow-color zone. The brom-thymol-blue test was recorded as negative when

*A project of the Haskell Animal Disease Research; from the department of bacteriology and hygiene, University of Delaware.

TABLE I—Results of examinations of samples from farm 1.

No. of Animal	No. of Exams	No. of Quarters	Group 1 Brom Thymol + Bact. Find. —	Group 2 Brom Thymol — Bact. Find. +	Group 3 Brom Thymol + Bact. Find. +	Group 4 Brom Thymol — Bact. Find. —
F-1	2	8		2	4	2
F-3	9	36	2	9	25	
F-4	2	8	1	3	1	3
F-5	7	26	4	7	1	14
F-6	11	44	2	14	13	15
F-7	4	16	5	1	2	8
F-8	6	24	5	3	6	10
F-9	10	40	7	10	11	12
F-10	15	60	4	4	12	40
F-11	14	56	7	19	24	6
F-12	14	56	7	23	16	10
F-13	2	8		3	1	4
F-14	8	32		16	11	5
F-15	3	12	2	5	4	1
F-16	5	20		15	3	2
F-17	4	16		4	1	11
F-18	3	6	1	1	2	2
F-19	3	12	1	4	7	
F-20	3	11			3	8
F-21	3	8		4		4
F-22	1	4			4	
Totals	127	503	48 (9.54%)	147 (29.22%)	151 (30.02%)	157 (31.21%)

the milk assumed a yellow-green or green-yellow color. A positive blood plate was one in which there were found alpha, alpha prime, beta, or gamma streptococci in appreciable numbers, hemolytic staphylococci, other organisms of mastitis significance, or any combination of these organisms. A negative blood plate was one in which the plate remained sterile or contained a few organisms of no mastitis significance.

The results of this study may be divided into four groups, as follows: 1) Samples which were brom-thymol-blue positive and blood-plate negative; 2) samples which were brom-thymol-blue negative and blood-plate positive; 3) samples which were brom-thymol-blue positive and blood-plate positive; and 4) samples which were brom-thymol-blue negative and blood-plate negative.

The samples from farm 1 were taken from 21 animals. There were 127 examinations made which represented 503 individual quarters. Of these samples, 48, or 9.54 per cent, fell into group 1; 147, or 29.22 per cent, in group 2; 151, or 30.02 per cent, in group 3; and 157, or 31.21 per cent, in group 4. This is shown in table I.

The samples from farm 2, shown in table II, were taken from 15 animals. There were 93 examinations made which represented 355 individual quarters. Of these samples, 28, or 7.88 per cent, belong to group 1; 85, or 23.94 per cent, to group 2; 173, or 48.73 per cent, to group 3; and 69, or 19.44 per cent, to group 4.

The samples from farm 3, shown in table III, were taken from 12 animals. There were 58 examinations made which represented 202 individual quarters. Of these samples, 10, or 4.95 per cent, fell into group 1; 79, or 39.1 per cent, in group 2; 105, or 51.98 per cent, in group 3; and 8, or 3.96 per cent, in group 4.

In table IV the results of the examinations of all the samples in the three herds are summarized. Of the 1,060 quarters examined, 86, or 8.11 per cent, fell into group 1; 311, or 29.34 per cent, group 2; 429, or 40.47 per cent, group 3; and 234, or 22.07 per cent, group 4. If groups 1 and 2 are considered together, in which there is disagreement between the two tests, there resulted 397 examinations, or

37.45 per cent, involving disagreement between the two tests. If groups 3 and 4, in which there is agreement in the two tests, are taken together, there were 663 examinations, or 62.54 per cent, which are in agreement.

As shown in table IV, there were 311 examinations in which the brom-thymol-blue test was negative and the bacteriological findings were positive. These contradictory results are not explained on the basis of a single type of bacterial infection but were found, as shown in table V, to be distributed throughout the several types of infection.

Of the 311 quarters examined in which the brom-thymol-blue test was negative and the blood plates contained significant growth, 101, or 32.48 per cent, contained alpha prime streptococci; 67, or 21.54 per cent, contained hemolytic staphylococci; 36, or 11.58 per cent, alpha streptococci; 32, or 10.29 per cent, beta streptococci; 10, or 3.21 per cent, mixed alpha and alpha prime streptococci; 10, or 3.21 per cent, gamma streptococci; 10, or 3.21 per cent, mixed hemolytic staphylococci and alpha streptococci; 7, or 2.25 per cent, nonhemolytic staphylococci; 6, or 1.93 per cent, mixed alpha and beta streptococci; 6, or 1.93 per

TABLE II—Results of examinations of samples from farm 2.

No. of Animal	No. of Exams	No. of Quarters	Group I	Group II	Group III	Group IV
			Brom Thymol + Bact. Find. —	Brom Thymol — Bact. Find. +	Brom Thymol + Bact. Find. +	Brom Thymol — Bact. Find. —
H-3	4	16	3	4	5	4
H-4	2	8		5		3
H-5	4	16	5		11	
H-7	7	25	1	16	1	7
H-8	11	35	4		30	1
H-9	14	55	3	4	48	
H-10	8	29	2	7	19	1
H-11	14	52	1	13	38	
H-12	13	52	3	16	13	20
H-13	7	28	1	12	2	13
H-14	3	12	3	1	1	7
H-17	2	8	1	2		5
H-18	2	8	1	4	1	2
H-19	1	7		1	3	3
H-20	1	4			1	3
Totals	93	355	28 (7.88%)	85 (23.94%)	173 (48.73%)	69 (19.44%)

TABLE III—Results of examinations of samples from farm 3.

No. of Animal	No. of Exams	No. of Quarters	Group I	Group 2	Group 3	Group 4
			Brom Thymol + Bact. Find. —	Brom Thymol — Bact. Find. +	Brom Thymol + Bact. Find. +	Brom Thymol — Bact. Find. —
W-12	7	28		15	13	
W-16	12	47	3	16	24	4
W-19	13	40		16	22	2
W-20	12	48	4	20	24	
W-27	2	2			2	
W-28	2	4			4	
W-29	2	7		1	6	
W-30	1	4		3	1	
W-33	1	4		2	2	
W-34	4	10	2	6	1	1
W-35	1	4	1		3	
W-36	1	4			3	1
Totals	58	202	10 (4.95%)	79 (39.10%)	105 (51.98%)	8 (3.96%)

cent, mixed alpha prime streptococci and nonhemolytic staphylococci; 5, or 1.61 per cent, unclassified streptococci; 4, or 1.29 per cent, mixed hemolytic and nonhemolytic staphylococci; and 13, or 4.18 per cent, were made up of *Escherichia coli, Pseudomonas aeroginosa,* or unidentified organisms.

TABLE IV—Grand totals of farms 1, 2 and 3.

	Number	Percentage
Quarters Examined	1,060	
Group 1: Brom thymol + Bact. find. —	86	8.11
Group 2: Brom thymol — Bact. find. +	311	29.34
Group 3: Brom thymol + Bact. find. +	429	40.47
Group 4: Brom thymol — Bact. find. —	234	22.07

Discussion

As the brom-thymol-blue test has been recommended widely as a field test in detecting mild chronic cases of mastitis, the investigation described was undertaken for the purpose of comparing the color reaction with brom-thymol-blue with the bacteriological findings in a group of selected clinical or subclinical cases of mastitis.

Milk samples were collected from individual quarters at weekly intervals from 48 milking cows, all of which had clinical or subclinical mastitis in one or more quarters of the udder. The brom-thymol-blue reaction was compared with the bacteriological findings of 1,060 individual quarters. In 663, or 62.54 per cent, of the examinations there was agreement between the two tests, whereas in 397, or 37.45 per cent, of the examinations there was disagreement between the two tests.

In 86, or 8.11 per cent, of the examinations the brom-thymol-blue test was positive when the bacteriological findings were negative. In 311, or 29.34 per cent, of the examinations the brom-thymol-blue test

TABLE V—Bacteriological findings. Group 2: Brom thymol—; bacteriological findings +.

Farm	1		2		3		All Farms	
Number of Quarters	147		85		79		311	
Percentage	47.26		27.33		25.40			
Mixed alpha and alpha prime streptococci	3	2.04	6	7.06	1	1.27	10	3.21
Alpha streptococci	14	9.52	17	20.00	5	6.32	36	11.58
Alpha prime streptococci	70	47.61	18	21.17	13	16.45	101	32.48
Beta streptococci	6	4.08	15	17.64	11	13.92	32	10.29
Gamma streptococci	4	2.72	6	7.06			10	3.21
Hemolytic staphylococci	33	22.44	9	10.58	25	31.64	67	21.54
Alpha and beta streptococci	5	3.40			1	1.27	6	1.93
Hemolytic staphylococci and alpha streptococci	4	2.72	3	3.53	3	3.80	10	3.21
Unclassified streptococci	4	2.72			1	1.27	5	1.61
Hemolytic and nonhemolytic staphylococci and alpha prime streptococci			1	1.18	3	3.80	4	1.29
Hemolytic and nonhemolytic staphylococci	1	0.68	1	1.18	2	2.53	4	1.29
Nonhemolytic staphylococci			3	3.53	4	5.06	7	2.25
Alpha prime streptococci and nonhemolytic staphylococci			1	1.18	5	6.32	6	1.93
Mixed staphylococci and streptococci				1.18	1	1.27	2	0.64
Pseudomonas	1	0.68					1	0.32
Mixed	2	1.36	1	1.18	1	1.27	4	1.29
Unclassified			3	3.53	3	3.80	6	1.93

was negative when the bacteriological findings were positive.

These results are in close agreement with those reported by Plastridge and Anderson.* They concluded from their studies that the brom-thymol-blue test is of no value in detecting apparently healthy carriers of organisms commonly associated with mastitis, that it is of slight value in detecting mild cases of mastitis, and that it may be expected to give color reactions indicative of mastitis in about 70 per cent of the tests when applied to samples from quarters which frequently give milk that is abnormal in appearance.

CONCLUSION

The brom-thymol-blue test failed to indicate mastitis in 29.34 per cent of milk samples from individual quarters which were positive upon bacteriological examination.

Aesculapius

Aesculapius, ancient Greek and Roman god of medicine, was a son of Hercules and the father of Polidarius of the Homeric heroes, and so on through the mythology of twelve generations of Grecian biographies down to the first Hippocrates, grandfather of the Great Hippocrates, famous father of medicine, who was a man, not a myth, born in the year 470 B. C.

Pharmacodynamics and Toxicity of Methenamine

Methenamine can be employed advantageously as a general antiseptic at the onset of infectious diseases but is not a specific for any one infection. Contrary to the prevailing opinions, however, it is not entirely inoffensive. It may cause hematuria and albuminuria, particularly where the kidneys are already lesioned. In dogs, especially adult and old ones, the dose should never exceed 0.03 Gm. (0.46 gr.) per kg. (2.2 lbs.). Young dogs will tolerate 0.5 Gm. (0.77 gr.) per kg., and these amounts should be given to obtain satisfactory results. Methenamine has no diuretic properties. In large doses it only provokes frequent micturition (pollakiuria) without augmenting the volume of urine eliminated. (*Dacheux. Thèse, Alfort. Abst., Recueil de Médecine Vétérinaire, cxv, May 1939, p. 307.*)

*Plastridge, W. N., and Anderson, E. O.: Storrs Agr. Exp. Sta. Bul. 184 (May 1933).

Safe Birdshot

Just because you missed all of those ducks on your last hunting trip is no reason the shot will do no damage, since many fowl die of lead poisoning after ingestion of shot in the course of feeding on heavily hunted ground. To prevent this unnecessary loss scientists at the University of Minnesota have perfected a shot which consists of lead and magnesium. The addition of magnesium does not interfere with the ballistic properties and contact of this alloy shot with moisture causes disintegration within 24 hours.—*Pathfinder*, December 16, 1939.

". . . . Where Angels Fear to Tread"

A popular weekly, widely circulated and quoted, sets the milker on the left side of the cow in a picture illustrating an advisory article on farming. Those who know milking all along the rough and rugged road of ovulation, insemination, fetal death, birthline accidents, puerperal infections, Bang's disease, bovine tuberculosis, mastitis, stable hygiene, feeds and feeding, butterfat values, bacterial counts, municipal regulations, coöperative selling, surplus milk and other sinister obstacles of the dairy farmer will not quarrel over sinister milking. Shifting the milker to the port side of the "ship" is wrong only by virtue of the fact that it just isn't done. Milkmaids from time-out-of-mind have chosen the starboard side and few cows take kindly to any change in this detail of milk production.

The possibility of left-side milking is not questioned. The point is that a bit more knowledge of such minor details would give the farmer a little more confidence in the endless flow of advice he receives from the sages of the urban centers.

The Sensitization of Cattle to Mammalian Tuberculin by an Avirulent Strain of Avian Tubercle Bacillus*

By JANET R. McCARTER, E. G. HASTINGS, and B. A. BEACH, D.V.M.
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EXPERIMENTAL evidence is substantially proving that sensitization by the avian tubercle bacillus is responsible for many nonspecific reactions of cattle to mammalian tuberculin in Wisconsin. Avian tubercle bacilli were isolated from the lymph nodes of two naturally infected cows of an experimental herd composed of 15 tuberculin-reacting cattle from various herds with no history of bovine tuberculosis,[1] and from one other tuberculin-reacting cow. After slaughter no gross lesions of tuberculosis were found in any of these animals. Also, avian tubercle bacilli were isolated from the lymph nodes of three of 21 tuberculin-reacting cattle in which lesions were observed during routine inspection at packing plants.[2] In none of these cases were bovine tubercle bacilli found in addition to the avian.

Two pure cultures of virulent avian tubercle bacilli and three mixed cultures of virulent avian tubercle bacilli and diphtheroids, inoculated subcutaneously, were previously found to sensitize cattle to mammalian tuberculin. This paper describes the identification of an avirulent avian tubercle bacillus isolated from a tuberculin-reacting cow, the tuberculin sensitization produced by this culture when inoculated into calves, and the pathogenesis by this culture in the calves.

EXPERIMENTAL PROCEDURE

The avirulent strain of avian tubercle bacillus with which this work was done was isolated from a composite of the lymph nodes of a cow from a herd with no history of bovine tuberculosis. This animal reacted to tuberculin but no gross lesions of tuberculosis were found on autopsy.

The cultural characteristics of this strain on coagulated whole egg with glycerin are similar to those of virulent avian strains in the amount and rate of growth but the consistency is more mucoid than butyrous and the color is light yellow. Growths on other mediums are also similar to those of virulent avian tubercle bacilli.

The result of the inoculation of experimental animals for the purposes of typing the culture and ascertaining its virulence is shown in table I. The inoculums were measured by weight and, to insure even suspensions, the cultures were ground in a tube with a fitted ground glass pestle attached to a motor.[3]

Although a large dose of the culture produced acute tuberculosis in rabbits soon after its isolation, chickens inoculated within a year after its isolation did not acquire tuberculosis. Even a dose of 10 mg. (.154 gr.) does not produce progressive tuberculosis in the rabbit several years after its isolation, although the culture does have some virulence, as shown by its growth in the rabbit that received 0.1 mg. and was killed after two months. (One one-hundredth mg. of virulent avian tubercle bacillus usually produces acute tuberculosis in the rabbit when inoculated intravenously.[4])

It might be supposed that this culture had been isolated as a mixture of avian tubercle bacilli and another less pathogenic acid-fast, since it produced fatal tuberculosis in rabbits soon after its isolation but became avirulent for rabbits after subculturing. Mixtures of diphtheroids and virulent avian tubercle bacilli have been isolated which, upon subculturing, "lost their virulence," *i.e.*, became pure cultures of diphtheroids.[5] However, this culture un-

*From the departments of agricultural bacteriology and veterinary science, University of Wisconsin. This work was supported in part by a grant from the Wisconsin Alumni Research Foundation.

TABLE I—Typing and tests for virulence on a culture of acid-fast bacteria isolated from a cow.

INOCULUM*	ANIMALS	TUBERCULIN TESTS† AND AUTOPSY RESULTS‡
Lymph-node composite from cow	2 rabbits 2 g. pigs	S 7 mos.; no tbc. S 7 mos.; no tbc.
Culture, 8 mos. after isolation; 5 mg.	2 rabbits 2 g. pigs	D 16 and 18 days; acute avian tbc. S 5 mos.; no tbc.
Culture, 12 mos. after isolation; 5 mg.	2 chickens	S 8 mos.; no tbc.
Culture, 5 years after isolation; 5 mg.	1 chicken	Tuberculin neg. after 4 mos.; D 5½ mos.; no tbc.
	1 chicken	Tuberculin neg. after 4 mos.; S 6 mos.; no tbc.; liver cultured; neg.
2 mg. intravenously	1 chicken	Tuberculin neg. after 4 mos.; S 6 mos.; no tbc.; liver cultured; neg.
	1 chicken	Tuberculin neg. after 4 mos.; S 11 mos.; no tbc.; liver spleen, lungs, and kidneys cultured; neg.; mic. no pathology liver, spleen, or lungs
15 mg. subcutaneously	1 chicken	Tuberculin positive after 4 mos.; S 6 mos.; no tbc. liver, spleen, lungs, and kidneys cultured; neg.
Culture, 7 yrs. after isolation; 10 mg.	1 rabbit	S 8½ mos. (gained 60 Gm.); 2 hard nodules 1 mm. in spleen; gross increase interlobular connective tissue in liver; no joint lesions; mic. neg.
1 mg.	1 rabbit	S 8½ mos. (gained 400 Gm.); no tbc.
0.1 mg.	1 rabbit	S 2 mos. (lost 40 Gm.); spleen 1 hard tubercle 3 mm. and 12 tubercles 1 mm.; mic. pos.; liver no gross tubercles; mic. pos.; lungs neg.
0.01 mg.	1 rabbit	S 8½ mos. (gained 590 Gm.); no tbc.

*Cultures used for inoculation were between 2 and 4 weeks old on autoclaved glycerin whole-egg medium. Rabbits were inoculated intravenously, guinea pigs subcutaneously, and chickens intraperitoneally, unless otherwise specified.

†The chickens used were all negative to avian tuberculin before inoculation.

‡S = Sacrificed; D = Died; tbc. = Tuberculosis; mic. = Microscopically. "No tbc." signifies that no gross lesions of tbc. were found upon autopsy, and that no acid-fast bacteria were found microscopically in smears from the spleen, liver, and lungs.

doubtedly has always been pure, and not a culture of avian tubercle bacilli mixed with a saprophytic acid-fast, since its cultural characteristics, as well as its pathogenicity for chickens, have remained constant following its isolation. Also, further evidence, when considered together with cultural characteristics and pathogenicity, is that serologically, by the precipitin test, the tuberculin protein from this culture can not be distinguished from the tuberculin proteins of virulent avian tubercle bacilli.[6] This tuberculin protein was obtained from the culture about five years after its isolation.

The culture was inoculated into calves to determine whether it would sensitize them to tuberculin and, thus, to make sure that it had sensitized the cow from which it was isolated. The results (table II) show that the avirulent avian tubercle bacillus sensitized two calves both to mammalian and avian tuberculins even though the culture was relatively avirulent, according to the findings at autopsy. The low virulence is indicated by the localization of the infection in the lymph nodes adjacent to the point of inoculation, by the fibrosis in the prescapular nodes, and by the failure to cultivate tubercle bacilli. (In our experience, the chances of isolating avian tubercle bacilli are greater by culture than by animal inoculation, even with fully virulent strains.[7]) The inoculation of 10 mg. of

TABLE II—Tests on calves for sensitization to tuberculin and pathogenicity with the avirulent avian tubercle bacillus.

INOCULUM	EXPERIMENTAL ANIMALS	TUBERCULIN TEST	RESULTS ON AUTOPSY
Culture, 6 years after isolation; 10 mg. subcutaneously in back	2 calves (about 3 months old)	(Intradermal) 3 days before inoc. both negative to mammalian and avian Old Tuberlin	S 3½ months, when in good health; inflammation and thickening at point of inoculation
		2 months after inoc. Calf 1: Mam. O.T.—Px6 diffuse Av. O.T.—Px3 diffuse Calf 2: Mam. O.T.—Px4 circumscribed Av. O.T.—Px2 diffuse	Prescapular lymph nodes nearest point of inoculation enlarged. No other gross lesion. Mesenteric, mediastinal, bronchial, and prescapular nodes cultured and examined histologically. No growth after 6 months in cultures. Mic. pathology negative except in prescapular nodes, which showed hyperplasia and fibrosis

virulent avian tubercle bacilli subcutaneously into calves may result in death within four to five weeks.[4]

The quantitative differences in the reactions to mammalian and avian tuberculins are of little significance, since many human beings react more to the avian than to the human tuberculin.[8]

SUMMARY

A culture of acid-fast bacteria isolated from a cow which had reacted to mammalian tuberculin has been shown by its pathogenicity for chickens, rabbits, guinea pigs, and calves; by its cultural characteristics; and by the behavior of its tuberculin protein in precipitin tests, to be an avirulent avian tubercle bacillus.

This culture sensitized calves to both mammalian and avian tuberculins, although the infection was localized in the prescapular lymph nodes nearest the point of inoculation, and tubercle bacilli could not be cultivated when the calves were slaughtered one month after the tuberculin tests. Microscopically, the only pathological findings were in the prescapular lymph nodes, which showed hyperplasia and fibrosis upon autopsy 3½ months after inoculation.

According to these findings, it might be expected that from tuberculin-reacting cattle sensitized by the avian tubercle bacillus, the sensitizing agent could not always be isolated, even though the animal were slaughtered shortly after giving the reaction.

These findings are in accord with those of Minett[9] in that avian tubercle bacilli of low virulence may be isolated from cows, although Minett's cultures were more virulent than ours.

In conclusion, it should be emphasized that even though bovine tuberculosis is being rapidly eradicated, cattle will continue to react to tuberculin until avian tuberculosis is also eradicated. The ratio of these nonspecific reactors sensitized by the avian tubercle bacillus to specific reactors sensitized by mammalian tubercle bacilli may be expected to continue to increase until a more concerted effort is made to eradicate the avian tuberculosis from the chickens and hogs in Wisconsin. This is an economic rather than a hygienic problem, since there is no evidence to show that avian tubercle bacilli are pathogenic for man under the living conditions in Wisconsin.

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Thrombosis of the Posterior Aorta

Thrombosis of the posterior aorta with its ramifications occurs in horses, cattle and dogs, although it is more frequently recognized in horses. The well-known symptoms of sudden onset after a given amount of exertion, sweating, manifestations of great pain, and pronounced lameness of the affected limb are not seen in dogs. Dogs so affected do not go lame. The manifestations are vacillating walk and, later, paraplegia. The feeble pulsation of the femoral artery of the affected limb does not facilitate the diagnosis as in the horse.

There is no effective treatment but the author suggests that possible benefit might be derived from periarterial sympathectomy. (*Daubard. Thèse, Lyon. Abst., Recueil de Médecine Vétérinaire, cxv, May 1939, p. 303.*)

I can assure you that men whose main job is planning our national defense believe that, if we make reasonable preparations, there isn't a power or combination of powers on earth that could threaten the Americas against our armed opposition.—*Gen. Hugh S. Johnson in Current History.*

Sprouted Grains

Prof. Oscar Elf of Ohio State University, well known in the veterinary profession for his important studies of nutrition in dairy cattle, is credited with having discovered that sprouted cereals tend to raise resistance to such chronic infections as tuberculosis, undulant fever and mastitis.

New Terminology of Diseases

Following the lead of the Thirteenth International Veterinary Congress, the Association created in 1938 a special committee on the nomenclature of animal diseases. P. J. Du Toit of the Union of South Africa heads the international committee and H. C. H. Kernkamp of University Farm, St. Paul, Minn., the A.V.M.A. committee. The purpose of these committees is to arrive at general agreements on the proper naming of the known diseases of animals in order to remove the confusion arising from the variety of names used in different countries and by different groups.

Mushrooms Devour Nematode Larvae

The predatory action of mushrooms on the larvae of the common roundworms affecting horses was studied as early as 1888 and at different times since that date. A recent research on the subject is that of Descazeaux (1939), who demonstrated that certain species of mushrooms will capture and digest the eggs and larvae of equine strongyles in a few days. The tests were made *in vitro*. Coprocultures in Petri dishes inoculated with mycelian filaments of *Arthrobotrys oliospora* and other mushroom species were completely sterilized. If numerous parthenogenetic females escaped the traps of the mycelia, they were, however, finally devoured along with the small larvae. The capacity of the mushroom to capture and lyse the larvae of equine nematodes appears to be unlimited. (*J. Descazeaux. Biologic sterilization of equine feces parasitized with nematode larvae [title translated]. Bulletin de l'Academie Vétérinaire de France, xii, April 1939, pp. 136-139.*)

The Relation of the Vitamin B Complex to the Nutrition of Domestic Animals*

By AARON ARNOLD,† Ph.D., and C. A. ELVEHJEM,‡ Ph.D.

THE RECENT applications of the laboratory findings on the dietary requirements and physiological rôle of the various members of the vitamin B complex to the nutrition of domestic animals makes it desirable to evaluate the data at this point. At the present time six distinct members of the vitamin B complex are recognized: Thiamin, nicotinic acid, riboflavin, vitamin B_6, pantothenic acid or the chick antidermatitis factor, and factor W. The severity of the pathological syndromes which result from a deficiency of these factors in the diet is no less dramatic than the cures which may be effected by the addition of the missing components. These laboratory studies are revealing and will further reveal applications as conditions which previously have not yielded readily to treatment or which are treated empirically are associated with the diseases produced by the nutritional approach.

THIAMIN

The fundamental finding of Lohmann and Schuster,[1] which established that thiamin pyrophosphate constitutes the coenzyme for the metabolism of pyruvic acid, demonstrated the physiological rôle of this factor in carbohydrate metabolism.

That thiamin is required preformed in the diet for rats, chicks, pigeons, dogs and man has been known for some time and requires no reiteration here. Karr[2] early demonstrated with dogs that anorexia is one of the beginning symptoms of a thiamin deficiency, in accordance with the previous observations on rats, chicks and pigeons.

More recently, evidence was presented that swine also require this factor in the diet. Foot *et al*[3] reported that two pigs fed a ration of rice, casein, salts and autoclaved yeast grew poorly and had convulsive fits accompanied by digestive disturbances, followed by death after five months on the thiamin-deficient diet. Two pigs fed the same regimen with the exception that the yeast had not been autoclaved showed normal growth gains for the duration of the 110-day experiment. Hughes[4] noted that swine fed a diet low in thiamin showed a tendency towards leg weakness along with a low growth rate. The addition of thiamin to the diet markedly aided the appetite and food utilization, which were reflected in a sharply improved growth rate.

The early investigations of Bechdel *et al*[5] conclusively demonstrated that the fermentations in the digestive tract of the ruminant supply a sufficient quantity of thiamin to satisfy the dietary requirement. Madsen *et al*[6] observed that sheep require an alcohol-soluble factor of the B complex which is supplied by yeast. A recent observation[48] indicated that horses may be susceptible to a deficiency of the B complex. On a diet of hay and oats, digestive disturbances resulted which were immediately alleviated by the administration of 250 Gm. (8.3 oz.) of dried yeast. It may not be amiss to record that our own studies[7] (submitted for publication December 1938) with dogs fed a baked dog-food product showed that fright disease appears to be associated with inadequate protein in the diet rather than a deficiency of thiamin, as has been reported.[8] Our studies with rats, chicks and dogs[9] further indicated that the dietary requirement for thiamin is approximately 1 part of thiamin chloride per million parts of non-fat, energy-supplying food materials for all species which require this factor.

In general, it may be expected that animals fed a sufficient quantity of untreated

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whole cereal grains will receive adequate amounts of this factor. Foods subjected to heat in the presence of moisture, such as tankage, dried whey, dried skim milk, and processed products, are likely to be low in this factor.[10] Grain by-products from which the germ and outer covering have been removed to improve the keeping quality are poor sources of the vitamin. Since anorexia, the primary symptom in the non-ruminating domestic animal, is not seen often in the field, it may be concluded that this factor is not likely to be deficient under field conditions.

NICOTINIC ACID

Warburg *et al*[11] and Schlenk and Euler[12] were among the first to demonstrate that nicotinic acid amide constitutes the prosthetic group in some of the coenzymes active in the respiratory systems in the body. Elvehjem *et al*[13] recently recognized that nicotinic acid and its physiologically related derivatives[14] will prevent and cure blacktongue in dogs, which is the manifestation of a dietary deficiency of this factor, as is pellagra in humans.

Miller and Rhoads[15] and Birch *et al*[16] observed severe disorders characterized by loss of appetite, anemia, dermatitis and diarrhea in swine fed a diet similar to the canine blacktongue-producing diet.[17] Chick *et al*[18] found that nicotinic acid alleviates this condition. The report[19] of the condition of the swine was similar to the syndrome as previously noted in dogs.[20] In the extreme stages of the deficiency all the pigs were emaciated and showed a rough, scurfy coat, with some dermatitis, particularly on the ears.

At autopsy little mesenteric or subcutaneous fat was present. The blood was watery and did not coagulate readily. The thin-walled hearts were flabby. In two of the pigs there were capillary hemorrhages in the mucous lining over the greater curvature of the stomach, duodenum and lower end of the ileum. There was extensive congestion and swelling of the mucous membrane at the ileocecal valve in the small intestine sometimes accompanied by shallow ulcers of irregular outline and considerable extent. The large intestine showed lesions of shallow ulcers near the ileocecal valve, spreading to a diffuse, necrotic enteritis extending over the whole mucous surface of the cecum and upper two thirds of the colon. The lymph glands on the lymphatics which drained the affected parts were swollen, edematous and often hemorrhagic. These disorders were prevented when the swine received 25 to 50 mg. (0.38 to 0.77 gr.) of nicotinic acid daily. Hughes[4] also noted that pigs fed nicotinic acid supplements were superior to those fed the unsupplemented basal diet of brewers' rice or rice screenings, casein, salts and cod liver oil with respect to thriftiness, hair coat, skin condition and general well-being.

Davis and Freeman[21] noted that necrotic enteritis in swine may be cured and prevented by feeding nicotinic acid. The infections are regarded as a secondary invasion associated with a low intake of nicotinic acid. Studies with 125 animals indicated that the cure in earlier cases is much more marked than in infections of long standing. Madison *et al*[22] observed anorexia, diarrhea and scurfy dermatitis in swine under field conditions which responded favorably after twelve days to the administration of 50 mg. of nicotinic acid daily.

Pearson *et al*[23] reported that lambs developed normally on a nicotinic acid-low ration. As is the situation with thiamin, the ruminant may not be dependent for this factor on an outside supply.

From a survey of literature we have concluded[24] that the daily requirement is 0.2 to 0.5 mg. (.003 to .007 gr.) of nicotinic acid per kg. (2.2 lbs.) of body weight.

From a consideration of the early data used for the experimental production of blacktongue in dogs,[19] it may be concluded that the grains and grasses are poor sources of nicotinic acid. Milk and wheat germ are also low in this factor.[25] The animal organs are an excellent source of nicotinic acid. The observations of Madison *et al*[22] are in accord with these views, since the swine were fed corn, oats, wheat middlings and skim milk. The swine also received variable amounts of tankage, which indi-

cates that this protein source can not always be relied upon to supply adequate nicotinic acid, due in part to the particular products contained therein and in part to the heat treatment accorded them. Since it is possible for this material to serve as a good source of protein and of nicotinic acid, it may some day be feasible to market this product on the basis of the biological value of its protein and its nicotinic acid content, in addition to its riboflavin content.

RIBOFLAVIN

Warburg[26] reported that riboflavin is the prosthetic group of the yellow oxidation enzyme, which is present in all living cells. Riboflavin has also been shown to be a component of a number of other enzymes. Hence, its importance for living tissue may well be realized.

Riboflavin is a dietary essential for rats, chicks, dogs and man, as shown by many investigators in the field. It is interesting to record that the nervous lesions accompanying a dietary deficiency of thiamin are now held to be a secondary effect[27] and that these lesions are actually due to an accompanying inadequate flavin intake.[28]

Lepkovsky and Jukes[29] reported that turkey poults developed a typical dermatitis as the result of a riboflavin deficiency. Sebrell *et al*[30] observed the following syndrome in dogs deprived of riboflavin: Bradycardia, cardiac arhythmia, yellow mottling of the liver, degenerative changes in the central nervous system, collapse and coma.

Hughes[4] found that swine fed a diet deficient in riboflavin gained slowly, frequently passed semi-fluid fecal matter, became crippled and walked with difficulty. Two of the pigs developed bowed front legs which turned out at the ankles and stood extremely close at the hocks. The angles of the hind legs were bent forward excessively.

Hauge and Carrick[31] were among the first to recognize that chicks require this heat-stable factor. This finding was confirmed and extended by the studies of Norris *et al*[32] and Record and Kennard.[33] It was observed that chicks fed a diet which contained yellow corn, wheat middlings or ground wheat, purified casein, minerals and cod liver oil grew poorly and developed a "curled toe" paralysis, which caused the chicks to walk on their hocks with the toes curled inward. Normal growth through 8 weeks of age resulted when the diet of the chicks was supplemented with 290 micrograms of riboflavin per 100 Gm. (3.3 oz.) of feed. It is interesting to note that while normal egg production resulted when the hens received 130 micrograms of riboflavin per 100 Gm. of diet, 230 micrograms of riboflavin per 100 Gm. of diet was necessary for normal hatchability of the eggs. Some good sources of this factor are dried whey or skim milk, alfalfa meal, yeast and liver. Since the level of flavin required for optimum performance is not likely to be supplied by grains alone, it is desirable that care be taken to insure an adequate intake of this factor.

VITAMIN B_6

While it has been demonstrated that several species of animals require vitamin B_6 preformed in the diet, no evidence has been obtained to indicate the enzymatic rôle of this factor. The recent elucidation of the structure[34] of the vitamin and its synthesis[35] will facilitate studies on its physiology.

Fouts *et al*[36] noted a deranged blood picture in dogs deprived of vitamin B_6. A similar syndrome was noted[37] in swine fed diets low in vitamin B_6. Early in the deficiency there was a small decrease in the number of blood corpuscles and a considerable decrease in size. At a later stage the number of corpuscles increased to about 50 per cent in excess of the usual number and their mean volume was reduced to one half. The hemoglobin level was half the normal, which thus constituted a microcytic hypochromic anemia.

In addition to the deranged blood picture there were severe nervous symptoms similar to those reported with dogs.[36] As the deficiency developed in the pigs, typical epileptic fits were observed which increased in frequency and severity as the experiment progressed. The pigs ran around, screamed and then dropped to the floor. Tonic spasms followed during which the legs were ex-

tended. This was followed by a clonic stage during which the limbs were jerked about violently accompanied by a grinding of the teeth and sometimes a urinary discharge. As a comatose stage set in, respiration was at first deep and stertorous, gradually becoming shallow. The duration of the fits was a few minutes to one quarter of an hour. Both the blood picture and the nervous symptoms were alleviated over a period of seven weeks by the administration of vitamin B_6 (eludate factor).

The requirement of the rat for this factor is 10 to 20 micrograms daily.[38] It is not possible to state at the present time whether the requirement of other species follows as a function of the body weight or of the metabolism.

Vitamin B_6 is supplied in ample amounts by the grains commonly fed under field conditions so that an adequate intake of this factor probably results under conditions of normal utilization.

PANTOTHENIC ACID, CHICK ANTIDERMATITIS FACTOR

Elvehjem *et al*[39] and Jukes[40] independently reported that pantothenic acid is probably identical with the factor which will prevent a condition in chicks characterized by incrustations around the eyes, the corners of the mouth, and on the legs and feet. In this condition the feathering is retarded. In addition, crusty scabs at the corners of the beak gradually enlarge and often involve the margins of the skin around the nostrils and underneath the lower mandible.

The deficiency of this factor in the diet of the chick is induced by heating grain rations dry at 120 to 130° C. for a period of 24 hours or more. Except in limited cases the grains fed under field conditions are not subjected to these heating conditions and, therefore, may be expected to supply adequate amounts of this factor. Lepkovsky *et al*[41] found no difference in egg production and hatchability when laying hens were fed a diet low in this factor.

FACTOR W

Lepkovsky *et al*,[42] Elvehjem *et al*,[43] Edgar and Macrae[44] and others showed that the addition of the previously mentioned factors of the B complex (with the possible exception of pantothenic acid) to purified diets for rats does not support growth unless the fraction remaining after the removal of these factors from yeast or liver preparations is also added. The factor, or factors, required for normal growth in addition to the aforementioned factors has been designated factor 2[42] (which is also meant to include pantothenic acid), factor W[43] or, simply, filtrate factor.[44]

Studies with swine disclosed[37] that serious difficulties arise under conditions of a limited intake of this factor. A moderate degree of anemia resulted, in accordance with the earlier observations of Wintrobe *et al*, on less complete diets,[45] in which both the blood corpuscles and the concentration of hemoglobin were reduced to about two thirds of their normal amounts. There was little change in the size of the corpuscles. Growth of the pigs fed the diet low in this factor was subnormal for six to seven weeks. Shortly before growth stopped it was noted that all of the pigs on experiment had weakness of the hind quarters and sagging of the back. The symptoms increased day by day and the gait became unusual, the hind quarters swinging as the animals walked. It appeared as if the back end were loosely attached to the rest of the body. The hind legs were slightly flexed at all three joints so that the posterior portion of the body was lower than the anterior. In another three weeks the hind quarters of two of the three pigs were almost completely paralyzed. The condition was a flaccid palsy and at no time spastic. The deep reflexes were absent and the pigs appeared insensitive over the distal half of their hind limbs. Addition to the diet of one pig of the missing component improved the general condition immediately, although the paresis of one leg, more severely affected, and clumsiness of movement did not improve.

Wintrobe *et al*[45] reported severe degeneration of the posterior columns of the spinal cord, the dorsal root-ganglion cells and the peripheral cells of pigs fed a diet

which supplied only 0.1 Gm. (1.5 gr.) of dried yeast per kg. of body weight per day. The pigs were given thiamin and, in the case of one pig, flavin supplements in addition. Under the conditions of the experiment it appears, therefore, that a deficiency of factor W was the first limiting factor, since no precautions were taken to insure adequate intakes of nicotinic acid, riboflavin (in the case of two of the animals) and vitamin B_6. The results indicate that swine are quite sensitive to a deficiency of factor W.

It seems probable that the posterior paralysis observed in swine by Biester and Eveleth[46] may be due to a low intake of this factor, as was observed by Chick *et al.*[37] and Wintrobe *et al.*[45] Concentrates of this factor are generally prepared from rice bran,[42] liver,[43] or yeast.[44] Thus, these materials are known to be good sources of the factor. Its distribution in other materials is not known as yet.

OTHER FACTORS

It may be pointed out in closing that evidence has been presented[47] to indicate that the B complex contains other factors which play a rôle in normal nutrition. Further study will be required to establish their places in the dietaries of species other than the ones which have been used to demonstrate their presence.

SUMMARY

The results of the studies on the vitamin B complex with rats, chicks and dogs are now being applied to the nutrition of domestic animals. It has been recognized for some time that the poultry industry has been materially benefited by these studies, particularly with regard to riboflavin. Field observations are substantiating laboratory results which indicated that swine require certain components of the B complex. It may soon be desirable to guard against a low intake of nicotinic acid and factor W. The earlier observations on the ruminant indicate that these animals are able to satisfy their requirements for at least some members of this group of dietary essentials by virtue of the fermentation which takes place in the digestive tract.

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Testosterone Deficiencies in Dogs

Experimental work has shown that the injection of estrogenic substances in males results in well-marked changes, such as prostatic enlargement and alterations in the mucous membranes of the genital tract, which are similar to those observed in females during estrum. The whole story of testosterone, which represents the secondary sex-characteristic-stimulating factor, is not entirely known, but one of its rôles is a neutralizing effect upon estrogenic substances. Hence, the use of testosterone seems indicated when there is a lack of this substance or an increased amount of estrogenic material in the system.

Three cases are cited in which testosterone propionate, a synthetic preparation possessing the same properties as natural testosterone, was used to good advantage. The first was a male Great Dane. The initial symptom noted was discomfort, with continual licking of the prepuce. An examination revealed a slightly swollen penis, inflammation of the mucous membranes of the meatus and a slight discharge. Local treatment, rest, vitamins and sulfanilamide were given, but without effect. The sheath became enormously enlarged and small ulcers appeared. A complete laboratory examination failed to reveal the presence of any pathogens. Finally, testosterone therapy was instituted. Five injections at four-day intervals were given. Noticeable improvement was observed on the second day. A reappearance several months later was promptly brought under control by two further injections.

The second case was an aged Irish Terrier. The previous history was a serious streptococcic infection and glycosuria. The condition under consideration was a continual discharge from the penis. The animal was uncomfortable and urination was difficult. The meatus was red and swollen, and the prostate somewhat enlarged but not tender. Treatment consisted of intramuscular injections of 5 mg. of testosterone propionate every five days. The symptoms entirely disappeared after the fourth dose. Sustaining doses were required at intervals, as the symptoms began to appear after about six weeks.

The last case was an aged Yorkshire Terrier. The prepuce was enlarged and edematous, with dark, reddened areas which suggested beginning necrosis. The penis was normal in size and the meatus purplish red. The discharge was whitish. Another symptom was a constant desire to urinate. The prostate was not hypertrophied. Testosterone propionate in 5-mg. doses was given at five-day intervals and, at the time the second dose was administered, decided improvement was noted. Because of a tendency toward recurrence, injections were continued at six-week intervals.

The authors conclude that these inflammatory processes are caused by the effect of the estrogenic substances in the presence of a deficiency of the neutralizing hormone testosterone. (*Etienne and Robertson. Canadian Journal of Comparative Pathology, iii, November 1939, pp. 301-305.*)

A Note on Echinoparyphium Recurvatum (von Linstow) Parasitic in California Turkeys

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ON AUTOPSY of a turkey from northern California in July of 1937, numerous flukes were found in the intestinal tract which were specimens of *Echinoparyphium recurvatum* (von Linstow).

One subject, a 10-week-old turkey, when autopsied showed a very severe inflammation of the intestinal mucosa with cecal involvements consisting of a pasty, cheese-like mass which greatly distended the organs. The cecal conditions were comparable in appearance with those found in typical cases of infectious enterohepatitis. No other changes of pathological significance were found. From the upper portion of the intestinal tract 267 adult flukes were recovered. Two younger turkeys (6 weeks old) from this lot did not show any pathological changes and were free of parasites.

A visit was made to the ranch on which they were raised. It is located near Wolf, Nevada county, Calif. The farm borders on a small creek from which the turkeys drink. An examination of the creek was made and two types of snails were collected. Two more turkeys were obtained for examination. Three turkeys were obtained from another ranch, about one-half mile up the creek. The latter birds proved to be free from flukes.

Laboratory examination of the snails collected proved them to be free from cercariae or metacercariae. Of the two turkeys brought back from the parasitized flock, one was examined at once and the other held in the laboratory for a few days. The first had 125 flukes in the intestinal tract. The mucosa did not show the severe inflammation previously seen, but there were a few large, well-defined hemorrhagic areas. The second turkey showed no evidence of intestinal inflammation, although a few flukes were found.

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THE PARASITE

E. recurvatum was originally described by von Linstow in 1873. Dietz (1909) redescribed the form and placed it in his genus Echinoparyphium. Tsuchimochi (1924) described *E. koidzumii* which he raised experimentally in domestic ducks. Yamaguti (1933) showed that *E. koidzumii* Tsuchimochi is a synonym for *E. recurvatum* (v. Linstow).

The author has compared the descriptions of *E. recurvatum* as given by Dietz, Tubangui, and Yamaguti, and Morishita's description of Tsuchimochi's species, *E. koidzumii*. The results show a great variation in the size of the parasite and its various organs. The measurements given below tend to increase the size range. However, methods of killing and fixing the material should be taken into account when comparing measurements.

The description is taken from specimens flattened between glass slides and fixed in 70 per cent alcohol. Two staining methods were used: Mayer's alcoholic cochineal, with or without an indulin counter stain, and Lynch's precipitated alum carmine.

Description.—The body is small and elongated. The anterior fourth is attenuated; the posterior three fourths have nearly parallel sides tapering slightly to the rounded posterior end. The parasite is 4.15-5.07 mm. long by 0.64-0.75 mm. wide. It is widest through the region of the acetabulum. The head collar is 0.35 mm. in transverse diameter. There are 45 cephalic collar spines, arranged in two alternating rows, with four large ventral collar spines on each side. The measurements for the spines are: Corner spines, 66-72.6μ × 14.3-15.8μ; aboral dorsal spines, 60-63μ ×

12.8-14μ; dorsal oral spines, 48.4 $\times$ 11μ; lateral spines, 55-57.2μ $\times$ 12.1μ. The cuticular spines extend from the cephalic collar to the acetabular region. The oral sucker measures 0.13-0.17 mm. $\times$ 0.13-0.14 mm.; the prepharynx is short; the muscular pharynx measures 0.11-0.14 mm. $\times$ 0.09-0.11 mm.; the esophagus is 0.48 mm. long. The acetabulum oval is 0.44-0.54 mm. in diameter.

The testes are elongated with smooth borders, tandem. The anterior testis measures 0.47-0.64 mm. $\times$ 0.21-0.35 mm.; the posterior testis, 0.56-0.71 mm. $\times$ 0.21-0.40 mm. The large cirrus sac is situated in front and to the right of the acetabulum. The genital pore is situated near the bifurcation of the gastric ceca. The ovary is median, oval, 0.074 mm. in diameter, between the anterior testis and acetabulum. Laurer's canal is present; the shell gland is larger than the ovary; vitellaria extracecal in pre-acetabular region, closely surrounding the testes and filling the intracecal space behind the posterior testis. The uterus is short and contains few eggs. The eggs measure 88-92μ $\times$ 60-64μ.

DISCUSSION

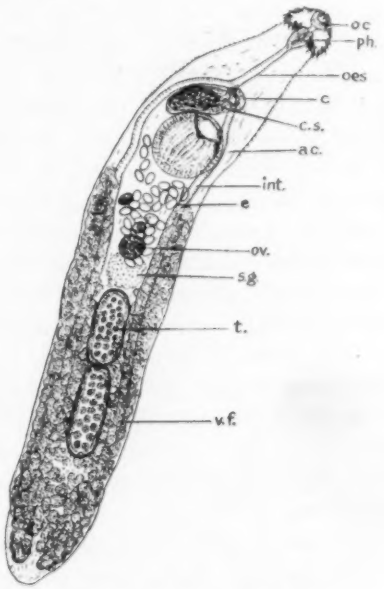
E. recurvatum has been found in the following hosts: *Anas platyrhynchos* (mallard duck), *Myroca marila* (greater scaup duck), *M. fuligula* (tufted duck), domestic ducks, chickens, and grebes. It has been found in Europe, Japan, Formosa, and the Philippine Islands. Its occurrence in turkeys is herein recorded for the first time, and its geographical range extended to North America.

According to Monnig, several species of snails have been found to act as first intermediate hosts. Bittner found that *Rana temporaria* acts as the secondary intermediate host, the cercariae encysting in the kidneys of the tadpole and the adult frog.

The creek to which the infested flock had access is only a few inches deep in most of its course, but here and there are large, deep pools protected by trees, undergrowth, and the necessary water plants which provide a situation attractive to the larger aquatic birds. Tadpoles were present in rather large numbers in the shallow water. Unfortunately, specimens were not obtained for laboratory examination.

With all the necessary stages for the fluke's development accounted for, it is quite probable that the mallard duck, or a related bird, constituted the original source of infestation in this outbreak.

An explanation of why the flock on the neighboring ranch, situated up the stream, was not infested may be explained on the assumption that the source of this outbreak started from a pool situated between the two ranches.



Echinoparyphium recurvatum, ventral view. ac., acetabulum; c, cirrus; c.s., cirrus sac; e, egg; int., intestine; oc, oral sucker; oes, esophagus; ov., ovary; ph., pharynx; s.g., shell gland; t., testis; v.f., vitelline follicle.

One other trematode of the family Echinostomidae was previously reported as occurring in turkeys. This species, *Echinostomum revolutum* (Froelich) may be separated from *Echinoparyphium recurvatum* by the number and arrangement of the collar spines, and by the long uterus filled with numerous eggs in the aforementioned species.

Echinostomes occur in domestic fowl more often than the literature reveals. Probably, a vast majority of infestations are never discovered. Numerous cases are observed but not recorded. A case of this

kind was called to the author's attention several years ago. These flukes may have been species of Echinostoma, since the chickens fed on snails and the cercaria of this species is known to encyst in snails. W. R. Hinshaw of the veterinary division of the University of California informed the writer that in 1931 the division received from northern California specimens of echinostome flukes found in a turkey. They were in such a poor state of preservation that a generic identification could not be made.

It is likely that flukes may be of great economic importance to the poultrymen situated near streams, lakes, or marsh-covered lands. Young birds seem to be especially prone to infestation. Death may occur in heavy infestation and is directly attributable to the flukes. Foggie recently reported such a case in young turkey poults from northern Ireland, in which the fluke was tentatively identified as *Plagiorchis laricola* (Skrjabin), normally a parasite of terns and gulls. He observed definite pathological changes in the intestinal mucosa. Smaller numbers of flukes may indirectly cause the ultimate death of the birds by lowering their resistance and subjecting them to disease.

Foggie found that treatment with Kamala (10 gr. per bird) was entirely ineffective in his case. Fencing, to prevent infestation, is the best solution for handling outbreaks of this nature and should be employed by all poultrymen whose flocks are apt to become abnormal hosts for parasites of our aquatic birds.

ACKNOWLEDGMENTS

The writer expresses his indebtedness to Emmet W. Price of the zoölogical division, U. S. bureau of animal industry, for his kind assistance in calling attention to important literature and to H. A. Hoffman, pathologist in the division of animal industry, California state department of agriculture, for his interest and encouragement during the study of this case.

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Sangamus Trachea

When chicks were hatched with hens and brooding was done in the barnyard, gapes was a veritable plague among the annual crop of young. The mortality sometimes ran as high as 100 per cent. With the coming of the commercial hatchery and brooder house, all this was changed. Gapes became a rare disease. In all of the literature on poultry diseases, reports of committees, poultry-congress programs, and lectures of recent years, one looks in vain for any mention of this onetime nemesis of the farm.

Sangamus trachea, the worm parasite that chokes the windpipe, has been robbed of the fertile field required to complete its life cycle. Walker (1886) considered the earthworm to be the intermediate vector and, 34 years later, Waite confirmed this observation. More recently, Taylor (1935) demonstrated that the larvae of this nematode can remain viable in slugs and snails for several years and, in 1938, Clapham (*Journal of Helminthology*, April 1939) reported that dipterous insects are also carriers.

Seventy-three per cent of American housewives prefer turkeys weighing under 13 pounds.

A Mouse Test for Measuring the Immunizing Potency of Antirabies Vaccines*

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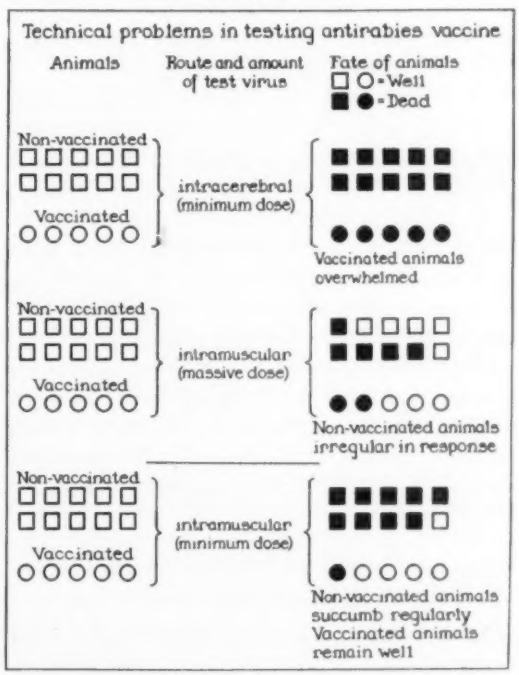
THE IMMUNIZING potency of a vaccine is tested usually by laboratory experiments followed by field demonstrations. In the absence of laboratory data, field statistics, such as comparisons of the relative incidence of rabies before and after the institution of a vaccination program, are usually not decisive as to the value of a vaccine. A controlled field experiment, on the other hand, is perhaps the ultimate test of the effectiveness of a given vaccine, but such a test is time-consuming. Consequently, it is usually prefaced by laboratory tests.

The crucial laboratory test of the potency of a rabies vaccine is the determination of whether or not vaccinated animals prove immune to the smallest dose of virus which is uniformly fatal to at least 50 per cent of unvaccinated animals when injected by a normal portal of entry. Such a test has not been easy to devise (chart 1). Experiment has shown that one can determine the smallest dose fatal consistently to 50 per cent or more of animals, that is, the minimal lethal dose, if the injection is made directly into the brain. Injection by this route, however, so lowers the resistance that both unvaccinated and vaccinated animals succumb. Thus, the effectiveness of the vaccine remains undetermined and the possibility exists that under more natural conditions of exposure, vaccination might have been successful. If, on the other hand, animals are injected with virus in a more natural manner, intramuscularly or through the bite of a rabid dog, their response is so irregular both in duplicate and in repeated tests that it is difficult to judge whether similarly exposed vaccinated animals survive because of the vaccine or uncontrolled variables.

STANDARD METHOD OF TESTING VACCINES NOT YET DEVELOPED

A review by the writer of experiments on the immunizing potency of antirabies vaccines[1] shows that no quantitative or standardized method of testing vaccines has yet been developed. Moreover, it is plain that the procedure of testing the immunity of vaccinated animals by injecting them intracerebrally with an unknown and considerable number of lethal doses of virus generally yields negative results. Finally, testing for immunity by injecting intramuscularly an unknown though massive number of lethal doses may demonstrate

CHART 1



*From the laboratories of The Rockefeller Institute for Medical Research. Presented at the 76th annual meeting of the A.V.M.A., Memphis, Tenn., August 28 to September 1, 1939.

at best a 50 per cent advantage of vaccinated over unvaccinated animals, provided the vaccine is administered in multiple doses approximating 1 per cent of the body weight.

Mouse Test Seems to Be Practical Quantitative Method for Determining Immunizing Potency of Antirabies Vaccines

The need for a quantitative practical test for determining the immunizing potency of antirabies vaccines seemed to be met by the development in our laboratory of a mouse test.[2] The test is carried out in the following manner.

1) Dilute the given vaccine tenfold, since the mice will not tolerate the amount of phenol or chloroform contained in the preparation as marketed.

2) Segregate 16 3-week-old Swiss mice for vaccination and 16 of the same age as controls. Provide five additional 2-week-old mice for the virulence test.

3) Inject the 2-week-old mice intracerebrally with 0.03 cc. of the diluted vaccine to determine the presence of virulent virus.

4) In the case of the canine vaccines, inject one eighth of the stated 5-cc. dose (0.6 cc.) intraperitoneally into each of the 16 3-week-old mice. Three weeks later test vaccinated plus control mice with two, four, eight, and 16 intramuscular doses of standardized virulent virus. The test virus is maintained by passing it regularly from brain to brain of 3-week-old Swiss mice. Full directions for its titration are given elsewhere.[2]

Results of Mouse Tests Carried Out with Commercial Vaccines

The following results were obtained on tests of five or more lots of canine vaccines, phenolized or chloroformized, from each of nine commercial firms, 50 preparations in all. None contained virulent virus. None immunized mice against a subsequent test intracerebral injection of as little as one lethal dose. When tested by the intramuscular method (table I), 27 phenolized preparations from seven manufacturers for the most part proved negative. Chloroformized preparations, on the other hand, especially from firms 1 and 2, have given results which merit further study.

The accompanying protocols illustrate the intramuscular potency test for canine vaccines and the type of result obtained. In experiment 4 (table II), all dilutions of virus proved fatal to nonvaccinated mice through 1:320. This was taken as the end point, although the titre may have been still higher. The No. 2 vaccine failed to immunize, whether given subcutaneously or intraperitoneally. The No. 1 vaccine likewise failed when given subcutaneously, whereas, given intraperitoneally in 0.6-cc. or 0.1-cc. amounts, it immunized against four and two lethal doses, respectively. The dose of 0.6 cc. corresponds to five times the 5-cc. dose advised for 10-kg. (22-lb.) dogs, while the 0.1-cc. mouse dose corresponds to the canine dose. Six-tenths cc. of the chloroformized vaccine given intraperitoneally appeared to irritate the mouse peritoneum. The animals seemed to be in pain for about an hour, were hyperirritable, and occasionally developed transitory convulsions. The discomfort, although causing loss of appetite for a day or two, seemed relatively harmless.

In experiment 5 (table III) vaccinated and nonvaccinated mice were tested against two strains of virus. One titred 1:320; the other, 1:640. No. 7 and No. 3 phenolized vaccines, plus No. 2 chloroformized vaccine, failed to immunize against more than two lethal doses. The No. 3 chloroformized vaccine, however, put up by the same firm that prepared the No. 3 phenolized product, protected mice against eight to 16 lethal doses when given in 0.6-cc. amounts and against two to eight when given in 0.1-cc doses.

In experiment 6 (table IV) the virus titred 1:1,280 in unvaccinated mice. No. 5, No. 6, and No. 8 phenolized vaccines failed to immunize, whereas No. 1 chloroformized vaccine, whether the 33⅓ per cent or the 20 per cent preparations, and whether 0.6 cc. or 0.1 cc. was injected,

TABLE I—Results of potency tests (intramuscular) of canine vaccines.

MANUFACTURER'S NUMBER	TYPE OF PREPARATION	NUMBER OF PREPARATIONS TESTED	AMOUNT OF IMMUNITY IN LETHAL DOSES
1	Chloroformized	7	8, 8, 2, 4, 4, 8, 8
3	Chloroformized	3	4, 16, 8
2	Chloroformized	4	4, 0, 2, 2
3	Phenolized	4	0, 4, 2, 2
4	Phenolized	4	0, 2, 0, 0
5	Phenolized	4	0, 0, 0, 0
6	Phenolized	4	0, 1, 0, 0
7	Phenolized	4	0, 0, 2, 2
8	Phenolized	3	0, 0, 0
9	Phenolized	4	2, 4, 0, 0
Total: 9 manufacturers		41 preparations	10 of 10 chloroformized vaccines from 2 manufacturers, positive; 3 of 4 chloroformized vaccines from 1, and 27 phenolized vaccines from 7 manufacturers, negative

immunized against at least eight lethal doses.

Taken together, the experiments show that no vaccine given subcutaneously and no phenolized vaccines given either subcutaneously or intraperitoneally immunized against more than two intramuscular doses of test virus. Chloroformized vaccines from two firms, however, immunized against four to ten doses when given intraperitoneally in amounts five to ten times those advocated for dogs per gram of body weight, and occasionally when given in amounts corresponding to the canine dose. The chloroformized vaccines, when given intraperitoneally in 0.6-cc. doses, caused transitory irritative phenomena.

MOUSE-TEST RESULTS REQUIRE CHECKING IN ANOTHER ANIMAL SPECIES

The mouse test as described above appears to be practical and to yield consistent results, yet these results still require checking in another animal species, preferably the dog. Our concern at this time is whether a canine vaccine which fails to immunize mice likewse fails to immunize dogs and, conversely, whether a vaccine plus a procedure which successfully immunizes mice will likewise immunize dogs.

Our experiments thus far suggest that the results of mouse and dog tests are closely parallel, but the work is still unfinished. The following experiments are submitted, therefore, as a progress report rather than a completed investigation.

The essential problem in the dog was the same as in the mouse, namely, to devise a technic of injecting virus into unvaccinated dogs which would be as natural as possible with respect to both route and dose and at the same time yield quantitative results.

PROCEDURE IN DOG TESTS

To reduce variability in response of individual dogs, a single breed, the Beagle, is largely employed. The animals are brought to the premises when about 4 months old. Here they are wormed, given 15 cc. (½ oz.) of anti-distemper serum, and maintained in quarantine pens on a standard diet. The test administration of rabies virus consists of injecting 0.25 cc. of virus, properly diluted, into the neck muscles of each side. A single strain of virus has been used thus far and passed routinely from mouse to dog and back to mouse. Following the injection the animals are taken to a different room and housed in separate cages where they are observed for two to five months. Animals found prostrate or dead are autopsied. Their brains are removed and a portion is inoculated intracerebrally into Swiss mice for identification of the virus.

TABLE II—Immunizing effect of canine antirabies vaccine on mice; comparison of effects of subcutaneous and intraperitoneal routes of injecting vaccine.

Mouse Group	Route of Vaccination	Fate of Mice Inoculated Intramuscularly with Virus in Dilutions					Amount of Immunity in Intramuscular Lethal Doses
		1:20	1:40	1:80	1:160	1:320	
A. No. 1 chloroformized 20% 0.6 cc.	i.p.	*8, 9, 21	9, 10, 25, S	10, S, S, S	S, S, S, S	—	4
B. No. 1 chloroformized 20% 0.1 cc.	i.p.	7, 8, 15, 29	8, 8, 11, S	8, 8, 10, S	8, S, S, S	—	2
C. No. 1 chloroformized 33 1/3% 0.6 cc.	i.p.	8, S	9, 9, S	8, 14, 16	S, S, S	—	2
D. No. 1 chloroformized 20% 0.6 cc.	subc.	8, 8, 12	7, 7, 10, S	8, 12, 13, S	9, 9, 10, S	—	0
E. No. 2 chloroformized 20% 0.6 cc.	i.p.	8, 8, 18	8, 8, 9	8, 9, 9, 13	11, 11, 13, S	—	0
F. No. 2 chloroformized 20% 0.1 cc.	i.p.	7, 8, 8, 8	8, 9, 9, 9	11, 11, 18, S	9, 10, 13, 15	—	0
G. No. 2 chloroformized 20% 0.6 cc.	subc.	7, 10, 15	8, 9, 9	8, 8, 13	8, 9, 11, 14	—	0
H. No vaccine		—	8, 8, 8	9, 15, 22	10, 10, 11, 15	8, 10, 22, 23	

* Day of death from rabies following test injection.
S = Mouse survived 40 days. — = Dilution not tested.

Results of Dog Tests

The response of unvaccinated dogs to this intramuscular injection is shown in table V.

In test P, three dogs given the 1:50 dilution succumbed. In test 1, six dogs given the 1:50 and three the 1:500 dilution succumbed. In test 2, four given the 1:50 dose died. In test 3, four given the 1:100 and four the 1:2,500 dilution succumbed, but only two of four given the 1:500 dose. In the fourth test, four given the 1:50, five the 1:500, and five the 1:10,000 all succumbed. In the fifth test, five given the 1:100 and in the sixth test five given the 1:200 dilution all succumbed. In the seventh test, six given the 1:200 dilution succumbed, but only two of five given the 1:4,000 and two of five given the 1:40,000 dilution died. Taken together the tests show that dilutions of 1:50 to 1:200, inclusive, were fatal to all of 37 dogs, and that 1:500 was fatal to ten of twelve. Dilutions of 1:2,500 to 1:10,000 were somewhat inconsistent in their effects and a dilution of 1:40,000 was fatal to less than 50 per cent, on a single trial.

These figures suggest that the minimum lethal dose for this virus for dogs intramuscularly lies between 1:500 and 1:10,000.

In table VI the durations of life from exposure to death of the test animals are grouped according to the dose of virus administered.

Seventy-nine per cent of animals given dilutions of 1:50 to 1:2,500 succumbed in 11 to 18 days, whereas only 26 per cent of those given the higher dilutions succumbed within this interval. About 20 per cent succumbed in 19 to 30 days, or after 30 days, regardless of the amount of virus employed. Finally, only 17 per cent remained well following an injection of 1:2,500 or more of the virus, in contrast to 47 per cent of those receiving higher dilutions.

These figures show that if 1:2,500 or more virus is given, 80 per cent of the animals respond in a relatively similar manner but that 20 per cent remain irregular regardless of the dilution of virus injected. They suggest, furthermore, that the minimum lethal dose is in the neighborhood of the 1:2,500 dilution.

This method of testing dogs is regarded, therefore, as relatively natural in that the

virus is introduced into the peripheral muscles and the animal responds after a ten-day incubation period with signs of typical dumb rabies. In the tests described below, the vaccinated animals received probably ten to 100 lethal doses, an amount by no means out of proportion to what they might receive through the bite of a rabid dog. The dogs are at least 80 per cent predictable in their response to this test. The shortcomings of the test are, first, that it is not carried out with strictly street virus. Titrations with street virus have thus far not been sufficiently consistent to be reliable. Second, the test dose, even though not excessive, should perhaps more nearly approach one lethal dose in order to determine a minimum amount of immunizing power possibly present in a weak vaccine. This shortcoming is of more theoretical than practical importance. All in all, the dog test, though by no means as critical as the mouse test, is sufficiently reliable to furnish the type of parallel evidence required.

The results of the above test exposure on batches of vaccinated and unvaccinated dogs are shown in the following protocols. At least 35 dogs are used in each test. Five to 15 of them are set aside as unvac-

TABLE III—Immunizing effects of canine antirabies vaccine on mice; comparison of chloroformized and phenolized vaccines.

Mouse Group	Test Virus	Fate of Mice Inoculated Intramuscularly with 0.01 cc. Virus in Dilutions							Amount of Immunity in Intramuscular Lethal Doses
		1:20	1:40	1:80	1:160	1:320	1:640	1:1,280	
A. No. 3 chloroformized 0.6 cc.	15811	*20, S, S, S	S, S, S, S	S, S, S, S	S, S, S, S	—	—	—	16
B. No. 3 chloroformized 0.1 cc.		9, 15, 20, S	20, S, S, S	14, S, S, S	18, S, S, S	—	—	—	2
C. No. 3 phenolized 0.6 cc.		8, 10, 11, 11	12, 15, S, S	15, 18, S, S	14, S, S, S	—	—	—	2
D. No. 3 phenolized 0.1 cc.		9, 18, 20, 20	17, 18, 18, S	12, 26, S, S	12, 15, S, S	—	—	—	2
E. No. 2 chloroformized 0.6 cc.		10,11,12,20	15, 17, 18, S	15, S, S, S	20, S, S, S	—	—	—	2
F. No. 2 chloroformized 0.1 cc.		12, 15, 19, S	10, 12, 15, S	10,17,18,27	16, 24, S, S	—	—	—	< 2
G. No. 7 phenolized 0.6 cc.		10,16,20,20	13, 18, 18, S	14, 23, S, S	S, S, S, S	—	—	—	2
H. No vaccine		10, 10, 12	9, 11, 13, 18	11, 17, 28	13, 14, S, S	10, 15, S, S	—	—	
A. No. 3 chloroformized 0.6 cc.	Sk.	—	9, 17, S, S	S, S, S, S	S, S, S, S	S, S, S, S	—	—	8
B. No. 3 chloroformized 0.1 cc.		—	12, 16, S	S, S, S, S	S, S, S, S	15, S, S, S	—	—	8
C. No. 3 phenolized 0.6 cc.		—	9, 17, S	9, 9, 9, S	11, 11, 15, S	10, S, S, S	—	—	2
D. No. 3 phenolized 0.1 cc.		—	7, 9	9, 9, S	9, 10, S, S	9, 10, 14, S	—	—	0
E. No. 2 chloroformized 0.6 cc.		—	7, 9, 15	9, S, S	29, S, S, S	28, S, S, S	—	—	2
F. No. 2 chloroformized 0.1 cc.		—	7, 9, 10	9, 10, 13	9, 10, 12, S	10, 12, 15, S	—	—	0
G. No. 7 phenolized 0.6 cc.		—	9, 10, 15	10, 10, S	12, 15, S, S	10, S, S, S	—	—	2
H. No vaccine		—	—	9, 9, 10, 11	9, 11, 12, 12	9, 9, 10, 10	9, 15, S, S	S, S, S, S	

*Day of death from rabies following test injection.
S = Mouse survived 40 days. — = Dilution not tested.

TABLE IV—Immunizing effects of canine antirabies vaccines; further comparison of chloroformized and phenolized vaccines.

Mouse Group	Fate of Mice Inoculated Intramuscularly with Virus in Dilutions						Amount of Immunity in Intramuscular Lethal Doses
	1:40	1:80	1:160	1:320	1:640	1:1,280	
A. No. 1 chloroformized 20% 0.6 cc.	*13, 14, 23	17, S, S, S	15, S, S, S	18, S, S, S	—	—	8
B. No. 1 chloroformized 20% 0.1 cc.	12, 12, 13	12,12,13,15	12,13,15,16	17, S, S, S	—	—	4
C. No. 1 chloroformized 33 1/3% 0.6 cc.	21, S	16, 20, S, S	14, S, S, S	12, S, S, S	—	—	8
D. No. 1 chloroformized 33 1/3% 0.1 cc.	11, 15, 17	12, 13, 14, S	17, 19, S, S	16, 17, 22, S	—	—	4
E. No. 5 phenolized 33 1/3% 0.6 cc.	—	12, 13, 17	13,14,15,17	14, 29, S, S	S, S, S, S	—	2
F. No. 6 phenolized 33 1/3% 0.6 cc.	—	13, 16, S	12, 22, S, S	12, 12, 20, S	12, 19, S, S	—	0
G. No. 8 phenolized 33 1/3% 0.6 cc.	—	13, 17, S	12, 16, S, S	12, 30, S, S	13, 17, 31, 2	—	0
H. No vaccine	13, 17, 25	12, 12, 30, S	—	12, 12, 17	—	15, 16, S, S	—

* Day of death from rabies following test injection.
S = Mouse survived 40 days. — = Dilution not tested.

cinated controls; the remainder are vaccinated, usually in batches of four or five. The canine vaccines are obtained through regular sources and the 5 cc. administered subcutaneously according to directions. Three weeks later the test injection is made with virus obtained from the brain of a mouse prostrate following its injection with virus-containing dog brain.

Table VII shows the results of tests of four phenolized vaccines and one chloroformized vaccine.

Both vaccinated and unvaccinated dogs were injected with the 1:100 dilution of virus. Five of five nonvaccinated dogs died in twelve to 18 days (100 per cent). Five of five dogs vaccinated with No. 4 phenolized vaccine died in nine to 26 days (100 per cent). Five of five dogs vaccinated with No. 5 vaccine died in twelve to 18 days (100 per cent). Four of five vaccinated with No. 8 phenolized and four of five with No. 9 phenolized vaccine died (80 per cent). One of four vaccinated with No. 1 chloroformized vaccine succumbed (25 per cent).

Clearly, none but the chloroformized vaccine had any immunizing effect on the dogs.

Table VIII shows the results of testing vaccinated dogs with a very small dose of virus, closely approximating one lethal dose.

Six of six dogs, unvaccinated, succumbed to the 1:200 dilution (100 per cent), as did two of three vaccinated with No. 3 phenolized and five of six vaccinated with No. 3 chloroformized vaccine, 66 and 84 per cent, respectively. Two of five unvaccinated and two of five vaccinated with No. 3 phenolized vaccine succumbed to the 1:4,000 dilution (40 per cent). Two of five unvaccinated dogs given the 1:40,000 dilution succumbed (40 per cent).

The vaccines in this test showed no immunizing effect, even against the minimum test dose of 1 to 4,000.

The final protocol (table IX) illustrates results of certain modifications in route and dosage of vaccines which are now under study. Four unvaccinated animals given 1:50; five, 1:500; and five, 1:10,000,

TABLE V—Intramuscular titrations of dog-passage virus, 15811, in 5-month-old Beagle dogs.

Test	Fate of Dogs Injected with 0.25 cc. of Virus into Neck Muscles of Right and Left Sides in Dilutions							
	1:50	1:100	1:200	1:500	1:2,500	1:4,000	1:10,000	1:40,000
P	*15, 16, 26							
1	14, 17, 17, 18, 22, 23			12, 14, 18				
2	11, 11, 11, 13							
3		12, 13, 14, 16		14, 18, S, S	17, 17, 17, 18			
4	13, 15, 15, 17			12, 13, 15, 27, 53			15, 18, 18, 23, 31	
5		12, 15, 17, 18, 18						
6			12, 13, 14, 23, 34					
7			14, 15, 18, 18, 21, 22			16, 23, S, S, S		22, 42, S, S, S
Total	17 of 17 (100%)	9 of 9 (100%)	11 of 11 (100%)	10 of 12 (83%)	4 of 4 (100%)	2 of 5 (40%)	5 of 5 (100%)	2 of 5 (40%)

* = Day of death from rabies following injection.
S = Animal remained well.

TABLE VI—Duration of life of Beagle dogs following injection of dog-passage rabies virus, 15811, into the neck muscles.

Dilution of Virus Injected	11 to 18 Days		19 to 30 Days		30+ Days		Remained Well	
	Proportion	%	Proportion	%	Proportion	%	Proportion	%
1:50	14 of 17	82	3 of 17	17	0	—	0	—
1:100	9 of 9	100	0	—	0	—	0	—
1:200	7 of 11	63	3 of 11	27	1 of 11	9	0	—
1:500	8 of 12	66	1 of 12	8	1 of 12	8	2 of 12	17
1:2,500	4 of 4	100	0	—	0	—	0	—
Totals	42 of 53	79	7 of 40	17	2 of 23	9	2 of 12	17
1:4,000 to 1:40,000	4 of 15	26	3 of 15	20	1 of 15	7	7 of 15	47

TABLE VII—Immunizing effects of canine antirabies vaccines on Beagle dogs (experiment 5).

Dog Group	Fate of Dogs Inoculated into the Neck Muscles (Right and Left) with 0.25 cc. of Dog-Passage Virus, 15811, Diluted 1:100		
	Day of Death Following Inoculation	Number Dead/ Number Injected	Per Cent Dead
A. No vaccine	12, 15, 17, 18, 18	5/5	100
B. Vaccine No. 4: Phenol, 5 cc. subc.	9, 10, 15, 18, 26	5/5	100
C. Vaccine No. 5: Phenol, 5 cc. subc.	12, 15, 16, 17, 18	5/5	100
D. Vaccine No. 8: Phenol, 5 cc. subc.	12, 13, 14, 19, S	4/5	80
E. Vaccine No. 9: Phenol 5 cc. subc.	12, 13, 14, 21, S	4/5	80
F. Vaccine No. 1: Chloroform, 5 cc. subc.	13, S, S, S	1/4	25

S = Animal remained well four months following injection.

TABLE VIII—Immunizing effects of canine antirabies vaccines on Beagle dogs (experiment 7).

Dog Group	Dilution of Test Virus	Fate of Dogs Inoculated into the Neck Muscles (Right and Left) with 0.25 cc. of Dog-Passage Virus, 15811		
		Day of Death Following Inoculation	Number Dead/ Number Injected	Per Cent Dead
A. No vaccine	1:200	14, 15, 18, 18, 21, 22	6/6	100
B. Vaccine No. 3: Phenol, 5 cc. subc.	1:200	12, 16, S	2/3	66
C. Vaccine No. 3: Chloroform, 5 cc. subc.	1:200	13, 16, 17, 21, 23, S	5/6	84
D. No vaccine	1:4,000	16, 23, S, S, S	2/5	40
E. Vaccine No. 3: Phenol, 5 cc. subc.	1:4,000	15, 18, S, S, S	2/5	40
F. No vaccine	1:40,000	22, 42, S, S, S	2/5	40

S = Animal remained well two months following injection.

TABLE IX—Immunizing effects of canine antirabies vaccines on Beagle dogs (experiment 4.)

Dog Group	Dilution of Test Virus	Fate of Dogs Inoculated into the Neck Muscles (Right and Left) with 0.25 cc. of Dog-Passage Virus, 15811		
		Day of Death Following Inoculation	Number Dead/ Number Injected	Per Cent Dead
A. No vaccine	1:50	13, 15, 15, 17	4/4	100
B. Vaccine No. 3: Chloroform, 1 dose, 20 cc., i.p.	1:50	34, 36, S, S, S	2/5	40
C. Vaccine No. 3: Chloroform, 1 dose, 10 cc., i.p.	1:50	S, S, S, S, S	0/5	0
D. Vaccine No. 3: Chloroform, 2 doses, 5 cc., i.p.	1:50	22, S, S, S	1/4	25
E. Vaccine No. 3: Chloroform, 1 dose, 10 cc., subc.	1:50	22, 23, 54, S, S	3/5	60
F. Vaccine No. 3: Chloroform, 2 doses, 5 cc., subc.	1:50	19, S, S, S, S	1/5	20
G. No vaccine	1:500	12, 13, 15, 27, 53	5/5	100
H. No vaccine	1:10,000	15, 18, 18, 23, 31	5/5	100

S = Animal remained well four months following injection.

all succumbed to the test virus. In contrast, of five given 20 cc. (⅔ oz.) of chloroformized vaccine intraperitoneally in one dose, only two succumbed; of five given 10 cc. (⅓ oz.) of the same vaccine intraperitoneally in one dose, none succumbed; 10 cc. in two doses of 5 cc., one of four succumbed. Of five given 10 cc. subcutaneously in one dose, three succumbed, and of five given two doses of 5 cc. subcutaneously, one only.

Taken together (table X) the tests show that of 34 unvaccinated dogs injected with virus in dilutions of 1:50 to 1:200, all succumbed (100 per cent) and of 34 vaccinated with phenolized vaccines from five different firms and injected as above, 30 died (88 per cent). Of 22 vaccinated with chloroformized vaccines from two firms and tested, ten succumbed (45 per cent). Finally, of eleven dogs vaccinated with 10 cc. of chloroformized vaccine intraperitoneally and tested, one died (9 per cent).

These phenolized vaccines, negative for dogs, were likewise negative according to the mouse test; the chloroformized vac-

cines, equivocal in dogs, were likewise equivocal in mice. The chloroformized vaccines in larger doses, given intraperitoneally, positive in dogs, were likewise

TABLE X—Summary of results on immunizing potency of canine vaccines (administered to dogs in one dose, 5 cc. subcutaneously; tested after three weeks with neck-muscle injections of 0.25 cc. of dog-passage virus, 1581I, diluted 1:50 to 1:200).

DOG GROUP	NUMBER DEAD/ NUMBER TESTED	PER CENT DEAD
No vaccine	34/34	100
Phenolized vaccine	30/34	88
Chloroformized vaccine	10/22	45
Chloroformized vaccine (10 cc. i.p.)	1/11	9

positive in mice. Thus, the results of dog tests parallel to date the results of mouse tests.

SUMMARY

We submit the mouse test as a practical, reliable measure of the immunizing potency of prophylactic vaccines for dogs and point out that this test has shown phenolized, single injection, canine vaccines to be lacking in immunizng potency. Further studies on the administration of chloroformized vaccines in larger amounts and by different routes, plus studies on still different types of vaccines, are in progress with a view to developing, if possible, an effective, specific method for the prophylactic immunization of animals against rabies.

References

[1]Webster, L. T.: The immunizing potency of antirabies vaccines. A critical review. Amer. Jour. Hyg., xxx (November 1939), No. 3, Sect. B, pp. 113-134.

[2]Webster, L. T.: A mouse test for measuring the immunizing potency of antirabies vaccines. Jour. Exp. Med., lxx (1939), 1, p. 87.

Farmers constitute 24 per cent of the American population but they receive only 11 per cent of the national income. Of this income, 11 per cent is derived from poultry.

Estrus Cycle of the Cow

The estrus cycle of the cow is quite regular. In 250 ovarian cycles of 38 cows, Strube observed that 80 per cent of these cycles had a duration of 17 to 23 days; 53 per cent, 19 to 21 days; 9 per cent, 24 to 28 days; and 4 per cent, 29 to 30 days. From these figures, it will be seen that the well-known 21 days (3 weeks) is quite regular. The exceptions are rare. Berthelon* divides the cycle as follows: Anestrus, 9 days; proestrus, 3 days; estrus, 24 to 36 hours†; and postestrus, 8 to 10 days.

Estrus (heat) may occur in cows without ovulation having taken place (Nielsen) but this is generally regarded as exceptional. According to McNutt (cited by Berthelon), ovulation in the cow occurs 24 to 65 hours after the beginning of estrus. Other authors give the moment of ovulation after the beginning of heat as follows: Hammond, 30 to 48 hours; Strodthoff, 18 to 27 hours; Kiriloff, 18 to 27 hours; and Berthelon, before the 24th hour.

In artificial insemination, Parchoutine and Vierevkine found that 18 hours after the very first manifestation of heat was the opportune moment to inject semen into the uterus. If injected after the 30th hour, the percentage of fecundations was low.

In the unbred or unfecundated cow, the involution of the corpus luteum is slow (23 days). The corpus luteum then disappears rapidly as a new one forms. The revolution of the estrual cycle is unique in the cow in that it is regular throughout the calendar year. The cycles succeed one another without interruption. When interrupted by fecundation, the cycles begin again 30 to 60 days after parturition. In rare cases (5 to 6 per cent) heat may occur during the early period of gestation (Nielsen).

The lightest particle of material is the electron.

*Recueil de Médecine Vétérinaire, cxv, February 1939.

†Applies to 90 per cent of cases. In 10 per cent the duration is shorter.

Lesions of Gossypol Poisoning in the Dog*

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A REVIEW of the literature reveals few references to the postmortem changes of animals presumably dying of cottonseed poisoning. There appears to be little doubt, however, that gossypol, the poisonous principle of cottonseed meal or cottonseed products, is toxic. It is also difficult to find references to symptoms encountered in cases of poisoning from this type of material. It is not the object of this paper to consider the symptoms or lesions applicable to all breeds of animals. Rather, the changes observed in several dogs fed on and dying while on a ration containing cottonseed meal will be discussed.

Healthy Foxhound puppies were placed on a diet containing 27 per cent by weight of cottonseed meal soon after weaning. The other components of the ration will not be mentioned here, since these will be discussed in other publications by Dr. Koehn of the department of animal nutrition. It may be stated, however, that the diet was adequate as regards essential factors, the cottonseed meal being used merely as a source of protein. The animals did not eat the ration readily at first but were induced to acquire a taste for it. They were fed all that they would consume, and near the termination of the experiment each was consuming approximately 240 Gm. (8 oz.) twice daily. Normal gains were made and the animals remained in apparent health for periods varying from seven to twelve months.

SYMPTOMS

There was posterior incoördination lasting for several days, followed in two or three days by stupor, somnolence, and lethargy, and terminating fatally within one to two days. Death ensued rapidly and the carcasses were in good condition.

GROSS PATHOLOGY

The dogs were autopsied soon after death. It was impossible in all cases to isolate pathogenic organisms from the blood, liver, spleen or various enriched mediums, and bacteria, parasites, or their larval forms could not be detected in stained blood smears. The possibility of other infections was precluded by the fact that dogs separated only by woven wire fences on both sides of this pen remained healthy.

No abnormalities of the skin were noted. Subcutaneous and visceral fat was abundant and, strangely, when compared to the fat of cattle on high cottonseed feed, was only slightly yellowish. The thoracic cavity contained, on the average, approximately one quart of a sanguineous fluid. There was extensive edema of the lungs and there were areas of ecchymotic hemorrhages scattered over the surfaces, with small pneumonic areas of the ventral apical lobes. The trachea was filled with a heavy, frothy, slightly blood-tinged mucous exudate. The heart was moderately hypertrophic, atonic, and edematous, and there were well-marked areas of degenerative changes of the myocardium. The valves were thickened by edematous infiltration and the pericardium moderately distended with a yellowish fluid. The blood was deep black and abnormally thick.

There was moderate ascites and the mesenteric lymph glands were hyperplastic, edematous, and congested. The liver was congested, moderately enlarged, friable, and studded with numerous areas of ecchymotic hemorrhages, indicating the initial stages of hypertrophic cirrhosis. The walls of the gall bladder were thickened, the mucous surface was covered with petechial and ecchymotic hemorrhages, and the bile

*From the department of pathology, Alabama Polytechnic Institute. Presented before the Section on Research at the 76th annual meeting of the A.V.M.A., Memphis, Tenn., August 28 to September 1, 1939.

was thick, ropy and flocculent. The spleen showed some evidence of fibrosis and the parietal surfaces were covered with large suggulations. The walls of the gastrointestinal tract were thickened and edematous. Numerous small erosions or ulcerations, 0.63 to 0.32 cm. (0.25 to 0.12 in.) in diameter, covered the pyloric portion of the stomach and the first 90 cm. (3 ft.) of the small intestine. The mucous surfaces were covered with a thick catarrhal-hemorrhagic exudate. The kidneys were slightly enlarged and the capsule peeled easily. The cortex was mottled and covered with areas of degeneration. The rectum of two of the dogs was filled with sand, probably indicating a perverted appetite. No abnormalities of the other organs were noted.

MICROSCOPIC PATHOLOGY

The tissues were fixed in Bouin's fluid, alcohol dehydrated, embedded in "Tissuemat," and sectioned at 6- to 7-μ (.000236 to .000275 in.) thickness. Hematoxylin-triosin stains were used. The brains were fixed in 95 per cent alcohol, embedded in celloidin, and sectioned at 15-μ (.00059 in.) thickness. Portions of each block were stained in 0.5 per cent aqueous tuloidin blue, and half in hematoxylin-triosin. Portions of the peripheral nerves were fixed in 10 per cent formalin, others in alcohol ammonia (Cajal's method). The formalin-fixed nerves were sectioned after 48 hours of fixation at 10-μ (.00039 in.) thickness with the freezing microtome and observed under the polarized-light microscope.

The axones of the alcohol-ammonia-fixed nerves were impregnated with silver nitrate, following Cajal's method, stained with ferric ammonium-sulfate hematoxylin and counterstained with triosin. The lungs showed extensive edema, both intra-alveolar and interalveolar, even though fixation to retain the fluid was not employed. Large areas of old and new hemorrhage were scattered over the entire sections, as well as numerous areas of emphysema of variable size. The majority of those alveoli not containing hemorrhage or edema were filled with a thick, mucofibrinous exudate. Marked granular and fatty degeneration of the myocardium and moderate edema both between and within the fibers appeared throughout the sections of the heart. There was a partial to a complete loss of the cross striations and there were numerous areas of degeneration of the fibers with leucocytic resorption. Several small to large areas of fibrous replacement of the myocardial fibers were scattered over the sections. Endothelial proliferation of the vessel walls appeared quite commonly.

Changes of hemorrhagic fibrous hepatitis were observed. Large areas of old and new hemorrhage were scattered over the sections. Marked fatty degeneration of the liver cords, areas of necrosis and several areas of fibrous replacement were seen. Fatty degeneration appeared more common toward the periphery of the lobules. Endothelial proliferation of the sinusoids occurred to a marked degree. The convoluted tubules of the kidneys showed considerable fatty degeneration with moderate intertubular hemorrhage of the cortex, and there were several areas of small, intratubular hemorrhage. There was no indication of hemoglobinuria in any of the cases.

Several areas of round cell infiltration and connective tissue replacement of the degenerated tubules were scattered over the cortex. A majority of the glomeruli were swollen and congested, the capsular space being filled with an inflammatory exudate, while a few were atrophic and sclerotic. There was a diffuse moderate sloughing of the tubular epithelium. The endothelial lining of the majority of the smaller vessel walls was proliferated and in several cases apparently formed a thrombus.

The abnormalities of the spleen were extensive areas of old and new hemorrhage and a slight increase in the size of the trabeculae, as compared with sections of the normal canine splenic tissue. The vessels of the spleen, in general, were constricted. The musculature of the gastrointestinal tract was edematous, with patchy areas of degeneration. Rather extensive areas of degeneration, sloughing, erosion or ulceration of the surface epithelium, as well as of the secreting tissue of the fundic portion of the stomach and the intestine,

were noted, the mucous surfaces being covered with a thick exudate composed of blood cells, desquamated epithelial cells and mucus. The ulcers extended deep into the submucosa and there was a rather heavy wall of fibrous tissue about their apices. No abnormalities of the other organs were noted.

Nerves observed under the polarized-light microscope showed rather large areas of demyelinization, the tissue being organized into various-sized oval, or round, fatty bodies lying between the sheaths and the axones. Many of the fibers were quite swollen at the nodes and along their courses. Fibers were, however, continuous and parallel. Fairly large areas of axonic fragmentation were observed to be distributed throughout the sections. The alcohol-ammonia-fixed nerves with the axones impregnated with reduced silver nitrate showed swelling, fragmentation and leucocytic resorption.

Changes within the central nervous system were not marked. They appeared only at the cortex of the cerebrum and at the junction of the mollecular and granular layers. None of the motor areas was affected. Sections stained with tuloidin blue showed a partial to more or less complete loss of the Nissl granules and swelling and distortion of the cells, some of which were of an ovoid or somewhat round form with eccentrically located nuclei. Primarily affected were the Purkingee and the large basket cells.

The majority of the blood-vessel walls were affected. The changes consisted of a thickening of the walls, especially of the intima, in which in several instances proliferation of the endothelial lining was evident. In a few of the cases perivascular cuffing was present to a mild degree. None of these changes was marked, all being mild but diffuse.

DISCUSSION

The extent of lesions found on autopsy in dogs fed for long periods of time on a ration containing 27 per cent cottonseed meal would never be suspected by the general appearance of the animal. After the animals acquired a tolerance to the taste of the ration, normal growth and development occurred. Symptoms appeared rapidly, were few in number, and not in any sense pathognomonic. While the lesions indicate a chronic course, death follows quickly after the appearance of the symptoms with a rather abrupt termination. The carcasses remain in good condition and show no indication of emaciation or dehydration. The only organs showing an acute change were the lungs, which were markedly edematous, with large areas of hemorrhage and pneumonic areas of the ventral apical lobes. Microscopic observation showed areas of hemorrhage undergoing organization and resorption, thus indicating previous changes. The other organs evidenced reparative changes as well as active degenerative processes. The toxic radical of cottonseed meal is apparently cumulative in action and some time is required before the lethal concentration is reached.

CONCLUSIONS

Gossypol, the active principle of cottonseed meal, is toxic and lethal for dogs. It exhibits an endotheliotoxic action. Extensive lung edema is probably the immediate cause of death.

Cottonseed meal in the amount of 27 per cent by weight of the ration over a long period of time is toxic and lethal for dogs.

False Pregnancy in the Bitch

The phenomenon of so-called false pregnancy in the bitch, which has been passed over as an inexplicable freak of reproductive physiology, appears to have been unmasked by E. J. Frick of Kansas State College, who demonstrated post mortem that the uterus of a bitch so affected is filled with a milky fluid. Bitches affected with false pregnancy pass through all of the signs of normal pregnancy, including a pronounced bulging of the abdomen that suddenly collapses at term without any apparent evacuation of the uterus. *(Editorial in The Jen-Sal Journal, xxiii, Sept.-Oct. 1939, p. 22.)*

Erythrocyte Sedimentation Studies in Dogs*

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SINCE Fahraeus[1] rediscovered, in 1917, that erythrocytes in blood samples from pregnant women settle out of suspension more rapidly than do cells from nonpregnant women, this phenomenon has attracted a great deal of attention. For some reason, however, this test is not used nearly as extensively in veterinary medicine as in human medicine.

Wharton and Wharton[2] reported an increased sedimentation rate in dogs with canine distemper but they did not give figures. No other references to this phenomenon in dog blood have been found.

In the production of serum for immunizing dogs against salmon poisoning, it was noticed that the red cells in citrated blood from sick dogs settled to the bottom of the container much more rapidly than did the cells from healthy, hyperimmune dogs. This observation led to some studies of sedimentation rates in dog blood. These were incidental to studies of salmon poisoning. Consequently, the number of tests made is small and the data are, in many instances, incomplete.

MATERIALS AND METHODS

Most of the dogs from which blood was obtained were being used in salmon-poisoning experiments. A few were kennel raised but the majority were obtained from pounds. They were usually mixed breeds or mongrels. They were kept in small outdoor pens with shelters for protection from inclement weather. Their rations consisted of a little milk, a small amount of raw beef or horse meat, and a commercial dog food. Dogs maintained in these quarters and on these rations remained in apparent good health as long as they were held, or up to four years.

Blood was collected in shell vials of approximately 30-cc. (1-oz.) capacity which had previously been marked at the 10-cc. (0.33-oz.) level with a glass etching pencil. Following sterilization of the vials, a label was attached to each in such a position that the top border of the label was at the etched line. This facilitated filling to the desired point and offered a convenient method of numbering the vials. As an anticoagulant, 0.1 cc. (1.5 minims) of sterile 50 per cent sodium citrate was placed in each vial shortly before it was used.

In a few instances blood was obtained through venapuncture of either the radial or the recurrent tarsal vein, an all-glass syringe being filled to the 10-cc. level and emptied immediately in the vial. In most cases, though, the more rapid and convenient cardiac puncture technic of Lockhart[3] was employed for collecting blood. In this procedure a 3-inch, 14-gauge needle was used. A snugly fitting piece of rubber tubing about 2 inches long was placed over the hub of the needle. The other end of the rubber tubing carried a piece of glass tubing about 1 inch long. After the heart was punctured and a few drops of blood had escaped, the free end of the glass tube was placed in the vial and blood was allowed to flow in until the 10-cc. mark was reached. A sterile rubber stopper was then placed in the vial and it was inverted a few times to insure thorough mixing of the blood and the sodium citrate solution.

Tests were usually begun within 10 to 20 minutes after the blood was collected, although in a few instances as much as an hour passed before the samples were set up.

After a few comparisons had proved that sedimentation rates in tubes of 18-mm. (0.7-in.) diameter and in Wintrobe tubes were approximately the same, the latter were used in all tests. These are glass tubes with inside diameters of 3 mm. (0.11 in.) and with flat inside bottoms. They are

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†Resigned from the staff of the Oregon station on May 1, 1938.

graduated in millimeter divisions to a height of 10 cm. (3.9 in.). By means of a pipette made for this purpose, the tubes were filled to the 10-cm. height and set up in a vertical position, the cotter-pin technic of Boerner and Flippin[4] being followed. Observations were usually made at intervals of five or ten minutes. Sedimentation rates were recorded on coördinate paper as graphs.

In some instances the volume of red cells in the specimens was determined through centrifuging the tubes after the sedimentation tests were completed.

SEDIMENTATION RATES IN NORMAL DOGS

Rates were determined in 20 blood samples from 15 apparently normal dogs. These animals varied in age from 8 weeks to full maturity. Both sexes were included. Since there were no apparent differences between rates of either the different ages or sexes, data on these factors are omitted from this report.

One of the animals had a rate of 12.5 mm. (0.49 in.) in an hour. Although an examination failed to reveal any indication of disease, it is probable that some abnormality existed, as blood from the same animal collected nine days later had a sedimentation rate of 2 mm. (.079 in.) in an hour.

The sedimentation rate of the other 18 samples was 1 to 4 mm. (.039 to .157 in.). There were six specimens with 1 mm., two with 1.5 mm., three with 2 mm., four with 3 mm., and three with 4 mm.

RATES IN PREGNANT BITCHES

Sedimentation rates were determined with 20 different blood samples from eight apparently healthy bitches which had been pregnant eleven to 56 days. The amount of sedimentation in these varied from 1 to 52 mm. (.039 to 2.04 in.), the mean being 15.4 mm. (0.6 in.). There were three samples, all from the same animal (No. 374), which were within what seemed to be the normal range (1 to 4 mm.). A marked increase above the apparent normal was observed as early as the 17th day of pregnancy. No uniformity in either the amount of the increase or the time at which it appeared was noted.

In five of the six pregnancies in which two or more tests were made, the rate increased as pregnancy advanced. In those in which the initial rate was low the subsequent increase was small, while a more rapid increase was recorded in those showing a relatively high rate at the first test. One animal (No. 374) was tested more than once during four different pregnancies. Marked differences were recorded in samples collected at approximately the same periods in these pregnancies.

RATES IN CANINE DISTEMPER

Sedimentation rates were determined with 21 blood samples from nine dogs affected with what was diagnosed as canine distemper. Since these animals were being held for later experiments, they were not under very careful supervision and, in most instances, symptoms were fairly well developed before the disease was detected. In no instance was a test made before the temperature rose. The amount of sedimentation recorded in an hour varied from 7 to 50 mm. (.27 to 1.96 in.), the mean being 26 mm. (1 in.) There was no apparent correlation between the amount of sedimentation and the height of the temperature. (See table I.)

Since Wharton and Wharton[2] reported anemia as a part of the syndrome of distemper and since it is well established that a decrease in the volume of packed red cells in blood is accompanied by an increase in the sedimentation rate, samples from some of the affected dogs were centrifuged. (See table I.) Dogs 758, 764 and 775 were puppies under 4 months of age, while No. 816 was a young mature dog. The packed-cell volumes in samples from these animals were nearly the same as those from normal dogs of the same ages which were being fed the same diets and which had sedimentation rates within the normal range.

RATES IN SALMON POISONING

A total of 38 tests with blood from 24 dogs with salmon poisoning were made. The amount of sedimentation in one hour

TABLE I—Sedimentation rates and temperatures of dogs with canine distemper.

Dog No.	Date	Sedimentation in Mm. in 1 Hour	Temperature	Remarks
758	4–9–35	23		
	4–11	50	103.6	
	4–17	26		Packed red cells, 35%
	4–20	38	104	
	4–24	20	104	
	5–1	33	104	Packed red cells, 32%
764	4–9	37		Packed red cells, 35%
771	4–8	17		
775	4–8	40	103	
	4–11	50	103.3	Packed red cells, 38%
	4–20	37	101.9	
	4–24	21		
	5–1	18	103	Packed red cells, 39%
777	4–8	40		
815	9–19	9	103.5	
816	10–9	8	104.6	
	10–17	20	101.6	
	10–31	7	100.4	
	11–8	4		Packed red cells, 44%; no clinical symptoms
828	11–8	13		
846b	3–6–36	12	104.2	

varied from 2 to 60 mm. (.079 to 2.36 in.), the mean being 25.8 mm. (1 in.). An increase in the rate was usually present as early as the fourth day following exposure, or shortly before the rise in temperature occurred. The tendency was for the amount of sedimentation to increase up to the twelfth day following exposure, but great differences among blood samples from different dogs collected at the same time interval after exposure were recorded. Three dogs which lived through attacks of salmon poisoning continued to have increased sedimentation rates until at least the 19th to the 21st days following exposure, or approximately a week after their temperatures had returned to normal and their appetites had been regained.

Discussion

It seems from the limited number of sedimentation tests reported in this paper that this procedure may be of value to the practitioner of canine medicine. The data indicate that an increased rate in a mated bitch which has a history of freedom from disease and which is showing no clinical symptoms may be considered as presumptive evidence of pregnancy. No information is available, however, on rates in pseudopregnancy.

If the minor digestive upsets with which the early stages of canine distemper are so frequently confused do not affect the rate of sedimentation, this test should prove of considerable value in the differential diagnosis of incipient distemper.

The low cost of the necessary equipment, the constant availability of blood for tests, and the small amount of time necessary for making a test will appeal to the busy clinician who is looking for diagnostic aids. Data submitted seem to justify further consideration of this procedure on the part of research workers and clinicians who are working with diseases of dogs.

Summary

In a limited number of tests the sedimentation rates with citrated blood from

Treatment with Sulfanilamide of Meningo-Encephalitis Associated with Canine Distemper

By M. L. MORRIS,* D.V.M., and T. J. MURRAY†
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MENINGO-ENCEPHALITIS associated with canine distemper has been considered for years as an incurable disease. The mortality is said to be 90 to 100 per cent. Goldberg and Volgenau[1] reported one recovery among 73 cases treated at the New York State Veterinary College. Our records indicate the mortality to be essentially 100 per cent when the disease is accurately diagnosed.

Canine distemper is an acute febrile, infectious, contagious disease caused by the filtrable virus of Carré. It is extremely devitalizing, since it has an affinity for the hemopoietic tissue. Infected dogs treated during the initial temperature rise (one to three days) with copious doses of hyperimmunized homologous serum usually respond satisfactorily. This early phase of the disease has passed, however, in the majority of dogs presented for treatment; the animal is entering the phase of secondary complications before it is possible to administer the specific serum treatment. In most instances, therefore, the veterinarian is forced to cope with the disease in its complicated form.

Meningo-encephalitis, commonly called nervous distemper, is a nervous syndrome following in the wake of canine distemper. Those experienced in the handling of canine distemper are cognizant of the fact that when the symptoms of meningo-encephalitis manifest themselves, the prognosis is unfavorable. As pointed out, the mortality has been essentially 100 per cent. Occasionally, a dog may live, but chorea, blindness, deafness, partial paralysis of the limbs, or other unsatisfactory sequels are usually present. In the past, attempts to treat the disease have resulted in the use of almost every drug employed in canine therapeutics. To quote Goldberg and Volgenau,[1] who treated 73 dogs: "Little could be done for those animals suffering from the nervous form. Many drugs were used with little or no result."

SPECIFIC ETIOLOGICAL AGENT OF MENINGO-ENCEPHALITIS IS UNKNOWN

The specific etiological agent of meningo-encephalitis is unknown. The disease in general has been looked upon as a sequel of canine distemper. It is generally agreed that canine distemper (uncomplicated) is caused by the virus of Carré. Some believe meningo-encephalitis to be a brain manifestation of canine distemper. However, Perdrau and Pugh[2] suggest that meningo-encephalitis is caused by a virus distinct from the virus of distemper. They state:

> On purely clinical grounds, therefore, one can not regard the encephalomyelitis under consideration as a specific sequel of distemper. * * *

*Raritan Hospital for Animals.
†Bureau of biological research, Rutgers University.

(*Continued from preceding page*)

normal dogs were from 1 to 4 mm. (.039 to .157 in.) in one hour.

Pregnant bitches usually, but not always, had rates considerably above the apparent normal.

Dogs affected with canine distemper had rates much higher than the apparent normal.

Rates were increased significantly in dogs with salmon poisoning.

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> For reasons just detailed it seems probable that the encephalomyelitis commonly referred to as the nervous form of distemper is not the result of the specific virus of distemper on the central nervous system * * *.

There may be a similarity between the "virus" of meningo-encephalitis in the dog and the virus of fox encephalitis in the red fox. Green and his associates[3, 4] have studied fox encephalitis extensively. They have been able to infect dogs with the virus of fox encephalitis by cisterna puncture, but not by intramuscular injection. Many dogs are refractory to inoculation. They are of the opinion that the dog is the natural reservoir of the fox-encephalitis virus. They state:

> We have found the dog highly susceptible to infection with the virus of fox encephalitis. The cellular inclusions typical of this virus were found in large numbers not only in the central nervous system but in the organs as well. It would appear from our studies that this virus must occur naturally in the dog. We have found whole litters of dogs highly resistant or highly susceptible to fox encephalitis, indicating a strong distribution of familial natural immunity.

Lockhart[5] and Lockhart and Johnson[6] described a diplococcus and a streptococcus as the cause of meningo-encephalitis. In our work we have been unable to culture any microörganisms under aerobic and anaerobic conditions from the brain and spinal fluid of dogs infected with meningo-encephalitis. We have been able, however, to culture streptococci and other microörganisms from the spleen and liver. (This is true also of dogs affected only with canine distemper without meningo-encephalitis.) It might be pointed out, however, that Green and his associates,[3] in their paper on the "General and Pathological Properties of the Fox-Encephalitis Virus," state:

> We have termed the etiological agent of epizoötic fox encephalitis a virus, as early in the course of our investigations infective material was found to be easily filtered, and cultures, for the most part, sterile. As our investigations have continued, these findings have become definitely established. The only culturable organisms which we have found associated with this infection at all frequently are streptococci, which often have been isolated in pure cultures from the brain of animals dying of the experimental infection * * *. Our more recent finding of an intranuclear inclusion typical of this infection definitely places the etiological agent in the group of filtrable or ultraviruses.

It is our belief in regard to the causative agent in meningo-encephalitis that, in some cases, the canine distemper virus has an action on the hemopoietic tissue which destroys the animal's protective mechanism sufficiently to permit the virus of Carré or an associated virus to attack the nervous system.

SULFANILAMIDE THERAPY

Since early in 1938, we have been treating meningo-encephalitis associated with canine distemper with sulfanilamide. The treatment was started as soon as the earliest nervous manifestations occurred. Based on the work of Marshall, Emerson and Cutting,[7, 8, 9] blood levels of sulfanilamide were determined during therapy. Green, Allison and Morris[10, 11] showed that the excretion of sulfanilamide in the urine of the dog is not constant.. The dosage of sulfanilamide, therefore, must be varied. The body weight of the animal can not be used as an accurate index of dosage but only as a guide. The sulfanilamide was administered in such dosage either by mouth or subcutaneously that the blood-sulfanilamide level was maintained at not less than 15 mg. (0.23 gr.) per cent.

It has been our policy in the treatment of canine distemper to study the blood pictures of dogs under treatment. Periodic total red and white cell counts and the Shilling differential white cell count were made. It can be stated briefly at this point that animals under treatment for canine distemper, which may develop meningo-encephalitis, show a lymphocytic leucopenia and a polymorphonuclear leucocytosis. The white cell picture is an excellent index for the prognosis of meningo-encephalitis, particularly when considered in conjunction with the various clinical symptoms.

To date, 30 animals affected with meningo-encephalitis have been treated with sulfanilamide. In the first study[12] we

TABLE I—Comparison of the blood pictures of normal dogs and dogs suffering from meningo-encephalitis before sulfanilamide therapy.

	2–8 Months				9 Months and Older			
	Normal		Meningo-Encephalitis		Normal		Meningo-Encephalitis	
	Average Number	Average Per Cent	Average Number	Average Per Cent	Average Number	Average Per Cent	Average Number	Average Per Cent
W. B. C.	12,165	——	15,364	——	11,467	——	17,912	——
Seg.	6,795	55.85	13,719	89.29	7,525	65.27	15,767	88.02
Lymph.	4,051	33.30	733	4.77	2,491	21.72	977	5.45
Stab.	816	6.70	422	2.75	753	6.56	646	3.61
Eosin.	484	3.97	316	2.06	623	5.43	262	1.46
Mono.	19	0.15	144	0.94	77	0.67	170	0.95
Juveniles	——	——	29	0.19	——	——	68	0.38
Ruptured	——	——	——	——	——	——	22	0.12

made, 14 dogs were treated, with 13 complete recoveries. This is a recovery of 93 per cent and can be compared to a mortality of 100 per cent in untreated animals. Of the 30 animals thus far treated, 23 have recovered. This is an approximate 77 per cent recovery. Of the seven animals that died, some were brought to the hospital in advanced stages of encephalitis and one was also infected with coccidiosis. Detailed data on each of the seven dogs that died are given in the appendix to this paper. It is our belief that only two dogs died that should have responded to the sulfanilamide therapy. This would indicate a recovery of 93 per cent, the same recovery rate as that previously reported.

In the animals which recovered there was a marked clinical improvement which usually took place in two to 14 days. Dogs with a total anorexia started to eat. Temperature returned to normal. There was an increase in the lymphocytes and a decrease in the polymorphonuclear leucocytes.

It should be emphasized that canine distemper is a complicated disease. It is essential that the clinician use careful judgment if satisfactory results are to be obtained. In addition to the meningo-encephalitis complication, the dog also may be suffering from a severe enteritis and diarrhea. Vomiting, fever, inappetence, avitaminosis and dehydration with rapid emaciation also may be present. Therefore, it is necessary, in addition to the use of sulfanilamide, that therapeutic procedures be instituted to correct these conditions. It is our practice to administer fluids of saline and dextrose with thiamin chloride in copious quantities. The diet must be carefully regulated. Constant temperature and good nursing are indispensable. Weak and debilitated patients are frequently given blood transfusions. In our experience, thus far, sulfanilamide is effective only for meningo-encephalitis. It will not arrest or benefit the other complications. It is therefore possible to treat a dog affected with canine distemper, meningo-encephalitis and other secondary factors with sulfanilamide and have death result. The sulfanilamide may be effective in arresting the meningo-encephalitis, yet the dog may die from one or more of the other complications.

Blood Picture

In table I is presented an average blood picture of normal dogs and dogs affected with meningo-encephalitis before they were given sulfanilamide. The average blood picture of normal dogs is taken from a report by Morris, Allison and Green (in press). There were 14 dogs suffering from the disease in the group 2-8 months old and 16 in the adult group. The average white blood-cell count is close to normal but it includes data from animals with a normal number of white cells, with a leu-

cocytosis and a leucopenia. There are, in fact, only two consistent changes in the Shilling differential leucocyte count which are found in the blood of all dogs diagnosed as having this disease, namely: An increase in the percentage of polymorphonuclear neutrophils and a decrease from normal in the percentage of lymphocytes. This increase in the percentage of polymorphonuclear neutrophils is due to an increase in the percentage of segmented forms. The stabs may remain normal, increase, or decrease, although the averages recorded in table I show a decrease from the normal. Usually, although not always, the percentage of eosinophils decreases.

The data recorded in table III were selected from some of the counts of individual dogs which were averaged in table I. They illustrate the different types of blood pictures encountered in this disease. The first counts, where the blood sulfanilamide is recorded as 0 mg. per cent, represent the distribution of white blood cells when the disease was diagnosed. Like all the others, these figures show an increase in the percentage of segments and a decrease in that of lymphocytes. These figures also show that with sulfanilamide therapy the blood

TABLE II—Group of dogs which died of meningo-encephalitis prior to 1938 before the use of sulfanilamide therapy.

	Young Group 15 Dogs		Older Group 8 Dogs	
	Average Number	Average Per Cent	Average Number	Average Per Cent
W. B. C.	11,180	—	11,975	—
Seg.	9,248	82.72	9,383	78.35
Stab.	1,049	9.38	1,560	13.03
Lymph.	715	6.39	727	6.07
Eosin.	106	0.95	141	1.18
Mono.	41	0.37	56	0.47
Basoph.	19	0.17	—	—
Ruptured	—	—	109	0.91

The data in table II were obtained from the records of 15 puppies and eight adult animals which died from meningo-encephalitis before sulfanilamide therapy was used. These data are similar to those recorded in table I, except for the average increase from normal of the percentage of stab cells. This difference in stab count may be due to the fact that the animals used to obtain the information for table II were in a more advanced stage of the disease than those considered in table I. It can be concluded, therefore, that the consistent and characteristic change in the Shilling blood picture of dogs suffering from meningo-encephalitis is the increase in the percentage of polymorphonuclear neutrophils and the decrease from normal in the percentage of lymphocytes. Other changes which are not consistent are probably attributable to variations within the disease or to secondary or accompanying conditions not always present.

picture approaches normal as the disease is brought under control. This response is considered to be a favorable one, especially since all of the dogs which died of this disease did not show it.

SUMMARY

Meningo-encephalitis is a common complication of canine distemper. The mortality is essentially 100 per cent when the disease is properly diagnosed.

The specific etiological agent is unknown. Some claim that the disease is due to a streptococcus and some believe that it is due to a specific filtrable virus. We have been unable to isolate any microörganism from the brain and spinal fluid of infected animals.

Thirty animals affected with meningo-encephalitis have been treated with sulfa-

TABLE III—Effect of sulfanilamide therapy on the blood picture of dogs suffering from meningo-encephalitis.

Date	W. B. C. 1,000's per Cmm.	Seg. %	Lymph. %	Stab. %	Eosin. %	Mono. %	Juv. %	Blood Sulfanilamide Mg. %
Dog 1244—Age 10 Months								
11–12–38	13.2	95	—	4	—	—	—	0.0
11–14–38	8.0	95	1	3	—	1	—	—
11–15–38	11.1	96	2	1	—	1	—	19.0
11–16–38	—	—	—	—	—	—	—	27.0
11–17–38	13.2	89	7	2	—	2	—	15.3
11–18–38	—	—	—	—	—	—	—	11.5
11–19–38	—	—	—	—	—	—	—	16.0
11–21–38	10.1	67	26	4	—	2	—	26.0
2–15–39	Still in good health							
Dog 1245—Age 10 Months								
11–12–38	8.7	92	4	2	—	1	—	0.0
11–14–38	10.3	93	4	1	1	1	—	—
11–15–38	6.8	89	5	3	3	—	—	16.0
11–16–38	—	—	—	—	—	—	—	12.5
11–17–38	8.2	85	8	2	3	1	—	25.0
11–18–38	—	—	—	—	—	—	—	16.5
11–19–38	—	—	—	—	—	—	—	17.5
11–21–38	10.5	74	20	5	—	1	—	17.5
2–15–39	Still in good health							
Dog 1234—Age 4 Months								
1– 4–39	19.0	92	4	3	—	1	—	0.0
1– 6–39	3.2	22	35	12	10	19	2	29.0
1– 7–39	3.8	41	26	14	6	10	3	—
1– 9–39	5.9	67	18	10	3	2	—	—
1– 9–39	Discharged with Rx sulfanilamide							
1–30–39	Returned for worming, appeared in normal condition							
Dog 1252—Age 1½ Years								
12–24–38	8.7	87	10	1	—	—	2	0.0
12–31–38	9.5	93	4	1	—	—	2	10.9
1– 2–39	8.9	68	30	1	—	1	—	18.5
1– 3–39	—	—	—	—	—	—	—	24.0
1– 4–39	—	—	—	—	—	—	—	14.5
1– 6–39	In good condition							
5–30–39	In good condition							
Dog 1243—Age 9 Months								
12–28–38	43.7	96	1	2	1	—	—	0.0
12–29–38	—	—	—	—	—	—	—	25.2
12–30–38	9.4	74	15	3	3	—	5	25.2
12–31–38	—	—	—	—	—	—	—	13.5
								16.5
1– 2–39	Dog discharged Rx sulfanilamide							
1– 6–39	In good condition, improving							
3– 6–39	In good condition, cured							

nilamide, with an estimated recovery of 93 per cent. In so far as possible, the blood-sulfanilamide level was maintained at not less than 15 mg. per cent. Sulfanilamide therapy alone is insufficient when other complications exist.

In the blood of all infected dogs there was an increase in the percentage of segmented polymorphonuclear neutrophils and a decrease in the percentage of lymphocytes. With sulfanilamide therapy, the blood picture approaches normal as the disease is brought under control.

ACKNOWLEDGMENT

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Appendix

Following are our observations on the seven dogs that died.

Dog 1251 was an inbred Irish Setter obtained from a breeding kennel. He was underweight, malnourished and did not gain weight when properly fed. This animal had been ill with canine distemper for at least two weeks before we started treatment. After 24 days he was destroyed, since the clinical evidence indicated that he would be left with serious after effects.

Dog 1250, a Beagle hound, was heavily infested with coccidia. Hound dogs are susceptible to distemper and infections involving the nervous system. Clinical experience indicates that dogs having distemper and heavy infestations of coccidia simultaneously, usually die, regardless of treatment. The blood picture in this case showed an excessive neutrophilic response but a persistent lymphopenia. Before death, sepsis occurred. Although fluids were administered, dehydration developed as a result of intestinal infection and the total white cell count rose to 40,000.

Dog 1258 was an aged Pomerian which through careful supervision had escaped distemper while young. Owing to the animal's age, the disease was misdiagnosed by the attending veterinarian. When presented to us the dog had been suffering with convulsions for several days and had been kept under the influence of barbiturates. We were requested to attempt treatment as a last hope. Extensive pathological changes were present before treatment was started.

Dog 575: Two months prior to treatment the tail of this animal was amputated. This was followed by incessant vomiting, for which serum was administered. Six days before the animal was brought to us he had nine convulsions. Marked pathological changes were manifested before the dog was presented for treatment. At the time of entry the animal was suffering from hemorrhagic enteritis, vomiting and convulsions. Death occurred 72 hours after treatment was started.

Dog 1255 was an inbred wire-haired Fox Terrier. This animal had been under treatment for three weeks and sick for several days prior to treatment. Generalized chorea had manifested itself several days before the animal was brought to us. Treatment in this case was instituted as an experimental effort; we held little hope of success.

Dog 1249 was an inbred English Setter female, nervous and neurotic. The animal had been immunized, elsewhere, with the Laidlaw-Dunkin method and was presented for treatment on February 9. She had dermatitis complicated with a temperature of 104° F. and there were slight changes in the blood picture. Copious doses of homologous anti-distemper serum were administered immediately. The animal was checked on February 14 and 17. During the eight-day period the blood picture showed only a moderate shift. By February 25, secondary complications began to manifest

themselves and the first typical blood picture was obtained. From that time on the dog developed a severe enteritis with fluid stools frequently streaked with blood. Tapeworm was present in the bowel. The patient also suffered from pneumonia. Although sulfanilamide treatment was administered at the beginning and followed through to the termination, satisfactory results were not obtained. An examination of a series of blood pictures of this animal showed that only transitory improvements occurred at any time.

We feel that the hemopoietic system was poorly developed and was overpowered by bacterial toxins and other complicating factors not directly associated with the cause of meningo-encephalitis. If this dog had been able successfully to combat these infections and intoxications, the sulfanilamide therapy would have controlled the meningo-encephalitis effectively, since no symptoms of this disease were displayed until March 22, one day prior to the destruction of the dog.

Dog 221 was a male Dachshund, one of a litter of five born to a dam by a sire, both of prize-winning stock. All five animals were given distemper virus and homologous serum. They were kept in the distemper ward for immunization. The virus used was possibly contaminated, since three of the five dogs dedeveloped symptoms of meningo-encephalitis. Sulfanilamide treatment was administered and two of the three recovered promptly. Dog 221 failed to show any response. We have no explanation to offer for the death of this animal. It may be pointed out that he was the smallest and most delicate of the litter. We definitely assign this dog a failure, even though he was treated promptly.

Paralysis from Rabies Vaccine

Notwithstanding the extensive use of rabies vaccine in dogs, postvaccinal paralysis is rare. Few authenticated cases have been reported. Because of the fact that paraplegia of various degrees is of frequent occurrence in dogs, it is not surprising that vaccination may be censured when that affliction attacks a vaccinated dog.

In human medicine, where rabies vaccine is given in repeated doses and is used only in exposed subjects, postvaccinal paralysis occurred in the proportion of 1 to 10,729 in a series of treatments recorded by the health division of the League of Nations. In another series the rate was 1 to 5,441 (*Jour. A.M.A.*, Nov. 4, 1939). It is unfortunate that similar observations are not of record for dogs, since the figures would surely be even more favorable from the standpoint of this paralytic sequel of rabies vaccination.

Paralysis following injections of rabies vaccine is supposed to be due to the vaccine itself, not to the virus or a combination of both. The belief that the paralysis is a manifestation of rabies partially arrested by the vaccine has not been supported, yet the fact that it occurs in bitten persons under, or soon after, treatment and practically never in thousands of unbitten but vaccinated dogs seems to indicate that the virus partially brought under control by vaccination may be the cause of the paralysis.

Rabies in Alabama

The Alabama state board of health, coöperating with the international health division of the Rockefeller Foundation, expended an appropriation of $108,000 on the investigation of rabies in that state from 1935 to 1938—a period of four years. The study of preventive measures under the compulsory vaccination law included observations of 220,000 vaccinated dogs, which is about one half of the entire dog population of Alabama. Of these, 134,000 were vaccinated in 1938.

In the laboratory work carried out in this extensive observation, attempts to cultivate street virus were not successful but a fixed strain of rabies virus was successfully carried through 68 passages. Growth could not be obtained on chick embryo. Rats could not be incriminated as factors in the epizoötiology of the disease. The final report of this prominent investigation will be of great importance to the veterinary profession.

Why male tortoise-shell-colored cats are so scarce, says the *Ford Home Almanac* (Oct. 1939), is one of the mysteries of nature that modern science has not yet solved. Females of that color are common but the male is so rare that in all England at the present time there are only seven known living specimens.

EDITORIAL

One truth dominates all history—it is easier to make war than peace. Everything can be made use of in waging war, even the vilest passions of the human soul—hatred, vanity, fear, vengeance, rivalry. To make peace one must possess difficult virtues—wisdom, moderation, justice, foresight.—Professor Guglielmo Ferrero.

Periodic Ophthalmia—A New Cause?

THE HISTORY of periodic ophthalmia dates back to antiquity but its etiology remains an unsolved mystery. Much has been said about diet, climate, filth, miasms, parasites and elevations above sea level as possible causes. In 1927, Rosenow reported that *Flavobacterium ophthalmiae* is the etiological factor. Shortly afterward, Woods and Burky denied Rosenow's work. About the same time, Woods and Chesney isolated a filtrable virus from a diseased eye which, when injected into the vitreous of healthy horses' eyes, produced a recurring uveitis resembling periodic ophthalmia. Yet, the infection did not invade the other eye and they were not able to reproduce the condition by any other means.

The disease has thus remained a virgin field for research. Perhaps, however, the interesting findings of its present investigators—Ophthalmologists Burky, Thompson and Zepp*—open a new byway to its etiology. Their stimulus was a 2-year-old filly suffering with a periodic ophthalmia in the right eye. This was the third attack and was associated with a profuse, yellow, mucoid discharge from the vagina. The trainer reported that such a discharge was always observed when the filly was in heat and that the period was always prolonged for 10 to 15 days. Examination by two veterinarians disclosed the presence of ovarian cysts.

Although this particular animal was not available for further study, the owner volunteered two aged mares for study who had developed periodic ophthalmia while the aforementioned filly was under observation. A detailed examination of these two animals revealed one positive finding—both were positive to the agglutination test for Brucella agglutinins. One of the mares was destroyed. A large cyst was observed in one of the ovaries and a strain of Brucella was isolated from the milk. Later, a blind gelding was autopsied and abscesses in the kidneys were noted. Smears obtained from these organs suggested Brucella but the cultures were negative.

A third animal, a 3-year-old filly with bilateral retinal detachment, was destroyed and a strain of Brucella was recovered from the ovaries. The agglutination test was negative. The ovaries were cystic and the spleen greatly enlarged. About this time, the second mare offered for study foaled a healthy colt and a strain of Brucella was isolated from the milk. In another case an autopsy of a completely blind mare revealed cystic ovaries, a slightly large and friable spleen and a liver studded with tubercle-like nodules. A cultural examination of the ovaries and blood revealed the presence of Brucella organisms. One mare that had a number of attacks showed an agglutination titre of 1:20 between attacks. Shortly after the eye became involved again, the titre would rise to as high as 1:80.

The authors cite an interesting case in which a farmer brought two pregnant sows to his farm. Both subsequently aborted. All of the animals on the farm were tested, with negative results. Shortly, one mare developed periodic ophthalmia for the first time. An examination of the eyes after the

*The rôle of Brucella in human ocular disease with special reference to periodic ophthalmia in horses. American Journal of Ophthalmology, Series 3, xxii (November 1939).

third attack revealed that no permanent damage had been done. The agglutination titre was 1:50. Two days after this examination the mare had another attack and the titre rose to 1:100. During the attack the animal was depressed, refused water and feed and the temperature was 101° F.

Mention is also made of a stock farm which raises Percheron horses. The hogs on this farm were infected with Brucella and drank from a stream that runs through the pasture used by the horses. Of approximately 70 mares on this farm, twelve were afflicted with periodic ophthalmia while not one of the ten stallions had any ocular disease. The stallions were isolated from the mares and swine, and water was obtained from an independent source.

Work with the strains of the Brucella organisms isolated from the mares produced ocular lesions in rabbits and guinea pigs. However, there was great variation in the lesions so produced by these injections. When the first strain isolated was injected into the anterior chamber of rabbits or intravenously into rabbits and guinea pigs, some of the animals exhibited ocular inflammation resembling periodic ophthalmia in horses.

The authors do not contend that periodic ophthalmia is due to this organism but, rather, that horses harbor it and that it, in turn, can cause ocular trouble in rabbits and guinea pigs. Yet, their findings suggest strongly that Brucella plays a part in this equine malady and definitely open a new avenue of approach to this perplexing problem.

Rather disconcerting, however, is the report of Thomas P. Cooper, who, speaking at the recent meeting in Chicago of the Horse and Mule Association of America, stated that agglutination tests were made on 52 horses with periodic ophthalmia and 69 normal horses, with positive results in 23 per cent of each group. In other words, Cooper apparently does not attach much significance to the findings of Burky and his associates.

Those "Good Old Days"

THE GOOD old days, meaning indefinite yesteryears, connote the time we got up in the morning to start a fire in the kitchen stove with wood brought in from the snow-covered woodpile; the time we chopped ice from the trough and thawed out the pump to water the horse; the time we drove snail pace over frozen mud roads that no brush can paint; the time of rattling-top buggies, cold feet, stinging hands and frost-bitten ears; the time when quacks were something to write home about; and the time before we had good colleges, good journals and friendly colleagues. Those *were* good old days because they led to days we should now enjoy with much less grumbling, with better grace, and with a greater sense of security. If we don't go static, 1940 will be good old days to the coming generations.

Diversified Farming

WHEN agriculturalists speak of diversified farming, they usually mean the rotation of the main crops—wheat, corn, oats, forage grasses, etc. But, says Christy Borth in his book, *Pioneers of Plenty*, many drugs now imported could be raised on American farms. Among these are belladonna, licorice, ephedrine, papain, gentian, rhubarb, camphor, opium, tragathanth, castor oil, and many others.

If the castor oil imported by the United States were produced at home, 160,000 acres of land would be needed for that purpose alone. Besides, the meal residue of castor oil production is a good fertilizer and soil insecticide and the sheath makes excellent string and rope. Moreover, the seed of the plant (*Ricinus communis*) contains a toxic insecticide principle worth about $4.00 a gram, yet to be exploited.

At the National Farm Chemuric Conference at Omaha in 1938, Perrin H. Long, a young scientist, pointed out numerous activities the American farmer is neglecting. A vast acreage, running into the millions that is now unfarmed or farmed without much profit, could be turned over to drug production to good advantage.

1939 A Banner Year for New Memberships

IN THE YEAR just ended 877 new members were enrolled in the Association as compared with 301 in 1938. Following are the men largely responsible for this record achievement.

Edwin J. Frick of Kansas State College, Manhattan, Kan., with 76 memberships to his credit, was the leader. Close behind were R. R. Dykstra of Kansas State College; H. D. Bergman of Iowa State College, Ames, Iowa; R. L. Mundhenk of Alabama Polytechnic Institute, Auburn, Ala.; Charles Murray of Iowa State College; C. F. Clark of East Lansing, Mich.; B. J. Killham of Michigan State College, East Lansing, Mich.; J. H. Knapp of Columbus, Ohio; Ross P. Marsteller of Texas A. & M. College, College Station, Texas; and E. E. Wegner of the State College of Washington, Pullman, Wash.

E. J. Frick

Excluding these men and President Way of New York City, who might be regarded as having considerable advantage, W. L. Curtis of Los Angeles, Calif., ranked first. Following Dr. Curtis were John H. Gillmann of Memphis, Tenn.; L. J. Allen of Oklahoma City, Okla.; J. E. Severin of Atlanta, Ga.; John Reardon of Galesburg, Ill.; Charles C. Rife of Atlanta, Ga.; and C. P. Zepp of New York City.

Resigned 1939

Blackberg, S. N., 333-11th Ave., Paterson, N. J.

Cass, Peter J., 417 Howard Ave., Burlingame, Calif.

Christie, Norman D., 493 Craig St., Winnipeg, Man.

Cook, Stanley C., 5804 Lathrop Place, College Hill, Cincinnati, Ohio.

Crider, Clayton L., Elkader, Iowa.

Cumming, William H., 1205 N. Morton St., Colfax, Wash.

Edwards, W. L., Box 567, Visalia, Calif.

Evangelista, Honorio C., 978 Bubbling Well Rd., Flat 2, Shanghai, China.

Graf, Charles J., Stuart, Iowa.

Gubser, Nicolas E., Earlham, Iowa.

Hadder, Walter J., Mecosta, Mich.

Hayes, Wm. F., Farmersville, Texas.

Herring, Lawrence J., Box 576, Wilson, N. Car.

Ivey, Wm. Edward, Apt. 1, 1025 Penn., St. Joseph, Mo.

Langdon, Harry B., Route 3, Charles Town, W. Va.

Leonhart, Oliver H., 1621 Birch, Oklahoma City, Okla.

Mann, Bernard R., 25 City Line Ave., Bala-Cynwyd, Pa.

Meyer, Joshua F., 1311 E. 15th St., Des Moines, Iowa.

Porter, Ray O., Box 767, San Luis Obispo, Calif.

Rathbun, Charles F., 543 Twelfth Ave. N., South St. Paul, Minn.

Redhead, William H., 3529 Broadview Rd., Brook Station, Cleveland, Ohio.

Thompson, Henry R., 1309 E. 32nd St., Kansas City, Mo.

Wheeler, N. M., Box 476, Winnsboro, Texas.

Youngberg, Stanton, Grove City, Ohio.

The Unification of the Profession

THE THOUGHT of solidifying the profession into a single unit is becoming quite universal. Inspired by the recommendations of the Committee on Public Relations and secretaries of component associations, one is prompted to predict that the general plan of organization adopted by the national medical and dental societies of this

country is the one toward which the veterinary profession will gradually drift in the next few years.

In principle, the plan is to join the membership of the national association through the local society, and through that portal only. Comparable with the county medical and dental societies, which serve as the door of admission to their respective national associations, we have many local bodies within the states to be used for that purpose. Many of them have the earmark of sufficient permanence to be bound to the state association in such a fashion as to make them the one and the only way of entering organized veterinary medicine.

A few years ago such a suggestion would have been thought far-fetched. To many eligibles, the A.V.M.A. has seemed too remote to be patronized. Whether the impression was deserved or not is less important than the fact that the interest of the members is now uppermost in mind and publishing a journal that will earn general approval by bringing useful reading material to the home and office with promptness and regularity once a month is the order for the future.

There seems to be considerable reason for predicting the early coming of a day when all eligible members and all branches of our profession have been molded into a trustworthy army strategically deployed and unafraid to assert the importance of the work it is delegated to do.

Correction

THE PRESIDENT of the Association was unaware that H. E. Biester of Ames, Iowa, who was appointed by Dr. Bergman to succeed the late Maurice Hall as A.V.M.A. representative to the National Research Council, is a three-year appointee. Dr. Way wishes to announce that W. W. Dimock of Lexington, Ky., who was appointed to succeed Dr. Biester, has graciously withdrawn his name in favor of the latter.

President Way deeply regrets this error, which is attributable to the fact that he did not consult Past-President Bergman and that there was no indication in the records of the duration of Dr. Biester's appointment.

The War and Cod Liver Oil

WITH THE normal movement of cod liver oil to America interrupted by the war abroad, the price of this commodity has increased considerably. We produce about 5 per cent of our requirements, which approach 5 million gallons a year. Ordinarily, about 60 per cent of our cod liver oil importations come from Norway and Iceland, 20 per cent from the United Kingdom and Germany and the remainder from Japan, Canada and Newfoundland. During the first ten months of last year, 6,225,000 gallons of cod liver oil were imported by the United States.

Another important development along this line is the relatively large import of fish livers from Japan. These livers are processed into medicinal oils and other closely allied products. Although the oil differs somewhat from cod liver oil, these products are competitive and thus may ease the demand for the cod liver oil.

As far as vitamin D is concerned, irradiated yeast can be employed effectively in feeds intended for four-footed animals. Apparently, however, irradiated yeast is deficient in the so-called "chick factor," its use thus being contraindicated in fowl.

Skim milk also has taken on added importance because of the war situation. This product is a common ingredient of most prepared feeds, since it contains vitamin B_1, the filtrate factor, and riboflavin or vitamin G.

It is therefore clearly apparent that purchasers of prepared feeds should buy from only reliable firms who have a reputation to maintain in spite of the hardships offered by the adverse effect on certain important feed ingredients.

The registration of donors for blood transfusions is being done on a large scale in England to prepare for the wounded soldiers. In the London area 250,000 have been typed and registered for that purpose.

Six years hence—1946—medical science can celebrate the centennial anniversary of the first surgical operation performed under profound anesthesia.

Man-Killing Scientists of War

MEDICAL SCIENCE is intended to save life but, when war breaks out nowadays, it is invoked to aid the gunners and bombers in the fine art of killing. The armies of certain countries have a germ-spreading service charged with the duty of causing diseases of man and animals. The veterinary service must not remain blind to this fact. History of the World War tells of the spreading of disease among domestic animals to hopple transportation and diminish the food supply of the enemy. As incredulous as this sort of service may seem to trusting pacifists, it is nevertheless true that germ warfare is a highly developed service, awaiting only the occasion to strike when, if and where the spreading of pathogenic organisms in one way or another will break down enemy resistance only. Medical science has put this new weapon in the hands of war departments that stoop to use them.

Since Alexander the Great, upstart of Macedonia, slaughtered certain urban populations to conquer the world, *c'est la guerre* has been the soldier's stock excuse for committing atrocities. But, in centuries to follow, Christianity tempered the thirst for blood. The killing was confined to the armed soldiers of the field. Then came the 20th century, when men of science took up the idea of making the cohorts of this ancient son of the Balkan country seem like philanthropic nomads. They propose wiping out whole populations with lethal gas and deadly contagions. Suffocation, pestilence and starvation have replaced the thrust of the spear.

It is doubtful whether any scientist, physician or veterinarian would admit being attached to such a service and, yet, someone more intelligent than the average soldier makes the plans and carries out the orders to spread typhus, typhoid, yellow fever, smallpox, cholera and bubonic plague among soldiers and civilians, and glanders, anthrax and foot-and-mouth disease among domestic animals in and out of the military service. They leave their fine souls behind and start out spreading instead of curing disease. Such is war in 1940, and there is no fairness in sanctifying one profession at the expense of another. A medical journal,* questioning the potentiality of germ warfare, blames everything upon "experts in the laboratory," as if the laboratory worker is less holy than his colleagues of the clinic.

The Women's Auxiliary

THE LONGER and deeper one studies the part that wives of veterinarians have played in the development of the veterinary profession, the more keenly one becomes aware of the failure of the national association to show its appreciation of the difficulties under which the Women's Auxiliary has labored in striving to build up a larger society than was possible under past arrangements.

It is a well-known fact that national societies in this country can not fulfil their purpose unless organized upon the basis of affiliated societies of states or districts as component units directed under central command. Nevertheless, the Women's Auxiliary of the American Veterinary Medical Association has done remarkably well, extraordinarily well, considering the floating membership with which an unbound national society has to contend.

As the central office has volunteered to aid the Auxiliary with its bookkeeping, correspondence and promotional work, it would be appreciated very much if the officers of our component associations in the states and provinces would urge at their meetings the formation of a formal ladies' auxiliary where none exists so that the general plan of reorganizing the national auxiliary may be placed before them for consideration and favorable action.

Needless to say, the present Auxiliary has been a godmother to many worthy students. Its intentions are to broaden the scope of its charity to the relief of worthy members in distress. The opportunity is unlimited, the effort worth while. All that is needed is the moral support of the doctors.

*Medical Times, lxv (Nov. 1937), page 550.

Code of Ethics of the American Veterinary Medical Association*

THE HONOR and dignity of our profession lies in our obedience to a just and reasonable code of ethics set forth as a guide to the members. The object of this code, however, is more far-reaching, for exemplary professional conduct not only upholds honor and dignity but also enlarges our sphere of usefulness, exalts our social standards and promotes the science we cultivate. Briefly stated, our code of ethics is the foundation of our individual and collective efforts. It is based upon the Golden Rule.

Section I

General Department

a) Conduct characterizing the personal behavior of a gentleman is expected of all members of the profession.

b) It is the solemn duty of all members of the Association to deport themselves in accordance with the spirit of this code.

c) This code is not intended to cover the entire field of veterinary medical ethics. Professional life is too complex to classify into a set of rules on duties and obligations to one's clients, colleagues, and fellow citizens.

Section II

Professional Department

a) No member shall use a college degree to which he is not entitled or any degree or title granted by an institution declared unworthy by contemporary institutions of its class.

b) No member shall belittle or injure the professional standing of another member of the profession or unnecessarily condemn the character of his professional acts.

c) Members shall comply with the common law governing their obligations to their clients and shall obey without obvious fault the official public regulations and laws governing their acts.

d) *Consultations.*—When a fellow practitioner or laboratory worker is called into consultation by the attending veterinarian, findings and discussions with the client shall be handled in such a manner as to avoid criticism of the attending veterinarian by his client.

In principle, a consultation should be conducted in such a manner as to display a spirit of professional coöperation of the highest degree between consultant and attendant, and thus improve the client's confidence in veterinary medicine.

Consultants shall not revisit the patient or client or communicate directly with the client without the knowledge and consent of the attendant veterinarian.

Laboratory workers in the rôle of consultants shall deport themselves in the same manner as fellow practitioners, whether they are private, commercial or public functionaries.

In no instance and under no circumstance shall a consultant take charge of a case or problem without the consent of all concerned, particularly when the client's financial obligations to the attending veterinarian have not been adjusted.

Section III

Advertising

a) Advertising as a means of obtaining patronage is objectionable in the practice of any branch of medicine. It is denounced as unethical and unprofessional. Veterinary medicine is not an exception. *Per contra*, on account of its widely misunderstood objectives, it is the branch of medical practice that is most vulnerable to fair and unfair criticism from other scientific pursuits.

Objectionable advertising consists of:

1) Advertising personal superiority over one's colleagues;

2) advertising secret remedies or exclusive methods;

3) advertising fixed fees for given services;

4) advertising as a corporation or partnership;

5) advertising case reports, allegedly unintentional;

6) advertising hospital and office equipment and the special service rendered therewith;

7) advertising the building or occupation of a new hospital as an unsolicited news item of the local press may be considered unavoidable and unobjectionable. Solicited and repeated publicity of this class is, however, frowned upon by the Association.

Directory Advertisements

a) Advertising in city, commercial, telephone or any widely circulated directory is a violation of this code.

b) A member who permits his name to be listed in directories in bold-face type or who advertises his name or hospital or institution in any way differing from the standard style, type or size used in the directory for the listing of professional groups (physicians, dentists, lawyers, nurses) is subject to the charge of unprofessional conduct.

c) It is also unethical for a veterinarian to allow his name to be printed in public directories as a specialist in the treatment of any disease or in the performance of any service within the scope of veterinary practice.

d) In principle, this section of the code of ethics is intended to improve the listing of

*The provisions of this document will be voted upon for adoption by the House of Representatives at the 77th annual meeting, Washington, D. C., August 26-30, 1940.

names in such a way as to give all of them identical visual prominence.

Advertising in Local Newspapers

a) It is customary and advisable in certain communities to print advertisements of professional men in local newspapers, and church, lodge and theater programs. But, such advertisements should be reasonable in size and display. They should be limited to name, title, address, office hours and telephone number.

b) Members are encouraged to write articles for the local press announcing the presence of contagious diseases and their seasonal prevention or treatment, provided the motive is a *bona fide* attempt to salvage the live stock of clients rather than personal gain. Wisely worded articles of this type add to the dignity and usefulness of the veterinary profession, whereas paid advertisements of the same subject are manifestly detrimental and, therefore, are violations of this code.

Advertising by Mail

a) The distribution of cards or circulars by mail or otherwise reminding clients that the time is at hand for rendering certain services (vaccinations, worm-parasite treatment, *et al*) is a questionable practice that should be frowned upon as objectionable advertising.

b) *Bona fide* personal letters or telephone calls of the same class as printed material may, however, be approved under special circumstances of grave emergencies, where professional dignity is not sacrificed.

Advertising by Personal Cards and Letterheads

a) The letterhead of a professional man should be modest, announcing only name, title, address, telephone number, and office hours.

b) In view of the turn veterinary practice has taken in recent years, a veterinarian may announce that he specializes in small animals or poultry on his cards and letterheads, provided that such cards or letterheads indicate that he is a member of the veterinary profession and thus distinguish him from groups of irregular practitioners who are not eligible to membership in the Association.

c) The mailing of letters or cards announcing a new location of office, hospital or other place of business is permissible. But, such occasions should not be used as an excuse for violating the code in other instances.

Advertising by Display Signs

a) Display signs of reasonable size and dimensions on veterinary hospitals are not regarded as objectionable, provided that they do not announce special services, such as bathing, plucking, clipping, x-ray work, etc., which characterize the ways of the charlatan.

b) Buildings, yards, and lots devoted to the treatment of animals, properly placarded as a guide to their location, are desirable evidence that a veterinary service exists in the community. In this respect, the veterinary profession may rationally claim to stand apart from other branches of medicine which denounce such displays as decidedly unethical.

Emergency Service

a) When called in an emergency as a substitute for a fellow practitioner in his absence, it is the veterinarian's duty to render the necessary service and then turn the patient over to his colleague upon his return.

b) In making emergency calls upon a patient already under treatment, it is unethical to institute radical changes in the treatment previously prescribed. Radical changes when deemed necessary should not be made in such a way as to attract unusual attention.

Testimonials — Guarantees — Frauds

a) Members of the Association shall not give testimonials as to the virtue of proprietary remedies unless such testimonials are based upon properly controlled scientific research conducted by the writer and reported in detail as such.

b) It is unethical to guarantee a cure by secret methods or otherwise.

c) Members of the Association shall avoid the impropriety of employing questionable methods to attract public attention or boast of possessing superior knowledge or skill in the treatment or prevention of any disease.

d) The issuing of false certificates of health on official documents is punishable by summary dismissal from the membership, and careless compliance with official regulations that the veterinarian is intrusted to enforce is deemed a violation of good professional behavior.

e) When employed by the buyer to inspect an animal for soundness, it is unethical to accept a fee from the seller. The acceptance of such a fee is *prima facie* evidence of fraud. On the other hand, it is deemed unethical to criticize unfairly an animal about to be sold. The veterinarian's duty in this connection is to be a just and honest referee.

Illegal Practice

a) It is unprofessional to aid in illegal practices of others.

b) Members of the Association shall not indulge in illegal practices, such as practicing without procuring a license or teaching others to do so in violation of the laws governing the practice of veterinary medicine.

c) It is the duty of members of the Association to report illegal practices to the proper authorities and to report such practices by members to the Executive Board.

Loyalty

The veterinarian should first of all be a good citizen and a leader in movements to advance community welfare. He should commit no act that will reflect unfavorably upon the worthiness of his profession.

APPLICATIONS

Rules Concerning Applications for Membership (Quoted from the by-laws of the Association):

Application for membership shall be made upon a blank furnished by the Association, in the handwriting of the applicant, and must be endorsed by two members of the Association in good standing, one of whom must be a resident of the state, province or territory in which the applicant resides. Application must be accompanied by the membership fee of $5.00 and dues pro rata for the balance of the fiscal year current, as stated on the application blank. Application must be filed with the secretary and be examined by him for correctness and completeness as far as available information will allow. After such approval by the secretary, the latter will cause to be published in the official JOURNAL, as soon thereafter as possible, said application with name and address of applicant, college and year of graduation, and names of vouchers. If no objections shall be filed with the secretary, as against the applicant's being admitted to membership in the Association, his name shall again be listed in the next issue of the JOURNAL, and if no objections shall have been filed within 30 days after the second publication of the name of the applicant, he shall automatically become a member and shall be so enrolled by the secretary, and membership card issued. If any objections be filed against any applicant, either on first or second notice, said application will be referred to the Executive Board for consideration.

First Listing

BAUMWELL, EARL
2233 Ocean Ave., Brooklyn, N. Y.
D.V.M., Ohio State University, 1938. Vouchers: C. P. Zepp and L. A. Merillat.

BORDER, CHARLES RICHARD
Elizabeth City, N. Car.
D.V.M., Ohio State University, 1938. Vouchers: Wm. Moore and A. A. Husman.

BREWER, N. RONALD
955 N. Hoyne Ave., Chicago, Ill.
D.V.M., Michigan State College, 1937; B.S., Michigan State College, 1930; Ph.D., University of Chicago, 1936. Vouchers: Edgar A. Crossman and L. A. Merillat.

COX, CHARLES EDWARD
Burgaw, N. Car.
D.V.M., Alabama Polytechnic Institute, 1936. Vouchers: Wm. Moore and A. A. Husman.

GOLDWASSER, HARRY I.
30-76 31st St., Astoria, L. I., N. Y.
D.V.M., Cornell University, 1935. Vouchers: W. A. Hagan and L. A. Merillat.

KELLER, WILLIAM G.
Constableville, N. Y.
D.V.M., Cornell University, 1930. Vouchers: Frank J. Baker and Almond H. Ide.

LENTNER, MAX SANFORD
Superior, Neb.
D.V.M., Kansas City Veterinary College, 1914. Vouchers: W. J. Moslander and Floyd Perrin.

MCCURDY, JON ALAN
Headquarters District "E" CCC, Camp Beauregard, La.
D.V.M., Iowa State College, 1938. Vouchers: T. S. Leith and W. T. Oglesby.

MCKINNEY, CLAUDE F.
Arthur, Ill.
D.V.M., Terre Haute Veterinary College, 1913. Vouchers: W. B. Holmes and L. A. Merillat.

MERCHANT, WILLARD R.
24 South 5th East, Salt Lake City, Utah.
D.V.M., Iowa State College, 1932. Vouchers: Jean C. Flint and R. E. Geisler.

NICHOLAS, DANIEL WEBSTER
Fairmont, Minn.
D.V.M., Chicago Veterinary College, 1916. Vouchers: Reuben B. Hovland and L. H. Phipps.

NIFFENEGGER, DEAN L.
Whiting, Iowa.
D.V.M., Iowa State College, 1939. Vouchers: A. H. Quin and E. B. Ingmand.

POTTER, HARLAND ROY
503 Drummond Rd., Stamford Centre, Ont.
B.V.Sc., Ontario Veterinary College, 1938. Vouchers: W. Moynihan and A. E. Cameron.

REDDIN, LESTER, JR.
163 Hunt Ave., Pearl River, N. Y.
V.M.D., University of Pennsylvania, 1937. Vouchers: A. Henry Craige, Jr., and Norman J. Pyle.

ROSEN, LOUIS E.
6966 Ogontz Ave., Philadelphia, Pa.
D.V.M., Alabama Polytechnic Institute, 1936. Vouchers: Robert R. Altaker and Roy F. Davenport.

SCHILLER, HARRY
66 W. Tremont Ave., Bronx, N. Y., N. Y.
D.V.M., Cornell University, 1938. Vouchers: James G. Catlett and L. A. Merillat.

SCHULTZ, R. R.
Mt. Vernon, Ind.
D.V.M., Terre Haute Veterinary College, 1914. Vouchers: J. C. Schoenlaub and L. A. Merillat.

STARKE, CHARLES FREDERICK
360 Northfield Ave., West Orange, N. J.
V.M.D., University of Pennsylvania, 1928. Vouchers: Arthur W. Smith and J. R. Porteus.

WESTERFIELD, CLIFFORD
Department of Animal Pathology, University of Kentucky, Lexington, Ky.
D.V.M., Michigan State College, 1938; B.S., Western Kentucky Teachers' College, 1930; M.S., University of Kentucky, 1932. Vouchers: F. E. Hull and W. W. Dimock.

WILSON, JAMES EWELL
1110 Gayley Ave., Westwood Village, Los Angeles, Calif.
D.V.M., Colorado State College, 1930. Vouchers: W. L. Curtis and E. B. Ingmand.

WIXOM, HERALD G.
250 E. Pine, Stockton, Calif.
D.V.M., Kansas State College, 1939. Vouchers: Edwin J. Frick and D. E. Settle.

Second Listing

Alkire, Robert Louis, Grantville, Md.

Badgley, Francis Roy, 39 Cedar Ave., Farmingdale, N. Y.

Bahensky, Melvin Donald, Palmer, Neb.

Barta, James Chester, 2916 S. E. 36th Ave., Portland, Ore.

Beadner, Harold, Box 44, Coquille, Ore.

Brooks, Willard C., Prairie Grove, Ark.

Broussard, George P., 417 E. Main St., New Iberia, La.

Brown, James Robert, 337 E. Washington St., New Castle, Pa.

Bulger, Edward Campbell, 34 Sanborn St., Lawrence, Mass.

Carr, Amasa Kenneth, 1378-13th St., San Pedro, Calif.

Cavenee, Waldo Robert, 204 S. Maple St., Fairfield, Iowa.

Clark, Marley C., 1514 Egleston Ave., Kalamazoo, Mich.

Cox, Orlando Fred, Mallard, Iowa.

Eisenhower, James M., 7970 Santa Monica Blvd., Los Angeles, Calif.

Foster, Edwin N., 1833 Cornaga Ave., Far Rockaway, New York. N. Y.

Hannahs, Morgan L., R. D. 2, Watertown, N. Y.

Holm, Glenn C., Bacteriology Department, University of Idaho, Moscow, Idaho.

Hurt, Ross Harrison, 1208 Maiden Lane, Pullman, Wash.

Ingle, Ronald Thomas, Department of Bacteriology, Ohio State University, Columbus, Ohio.

Jacobi, Van S., 1001 N. 4th St., Watertown, Wis.

Jones, L. Meyer, Department of Veterinary Physiology and Pharmacology, Iowa State College, Ames, Iowa.

Kahl, Bernard D., 299 Central Park Avenue, Yonkers, N. Y.

Kellman, Carl, 1875 Carter Ave., Bronx, N. Y.

Lammey, Claude L., 555 Hill St., York, Pa.

Lee, Philip Arthur, 236 Elm St., Reno, Nev.

Livengood, Abraham G., Salisbury, Pa.

Lukens, William R., 211 W. Main St., Hillsboro, Ohio.

McDole, Donald Harris, 9088 Santa Monica Blvd., Los Angeles, Calif.

Meyer, George Bernard, South Main St., Washington, R. I.

Moul, Evan L., 286 Pleasant St., Concord, N. H.

Moxley, Elmer Darwin, 940 N. Highland Ave., Los Angeles, Calif.

Nathanson, Sidney, 320 Church Ave., Brooklyn, N. Y.

Naylor, John Richard, 8018 E. Colfax Ave., Denver, Colo.

Pfaffman, G. A., P. O. Box 175, Woodland, Calif.

Reneau, Jr., John B., Box 457, Paducah, Texas.

Sargent, Andy John, 1151 N. Highland Ave., Hollywood, Calif.

Scott, Laurance Palmer, 1603 W. 4th St., Waterloo, Iowa.

Weston, Ralph Silas, 322 San Anselmo Ave., San Anselmo, Calif.

Williams, Fred Woods, 1240 Montgomery St., Oroville, Calif.

Williams, Harvey C., Route 1, Box 71, Clarksburg, W. Va.

Wright, Carlton J., Cerro Gordo, Ill.

Zweig, Harry M., Nassau, N. Y.

CLINICAL DATA

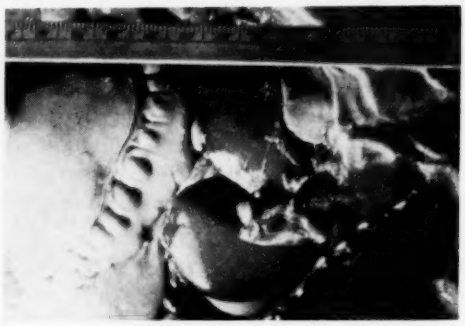

Fig. 1. Liver and spleen of the affected beaver. Note the pin-point lesions.

Tularemia in a Beaver*

By H. L. HAMMERSLAND and E. M. JONESCHILD, D.V.M.
Helena, Mont.

IN 1912, McCoy and Chapin reported the discovery of the causative organism of tularemia and since that time the disease has been reported in man and various species of animals and birds. On previous occasions this laboratory has reported the disease in blue grouse, sage hens, sheep and rabbits. It is our understanding that the disease has been reported from Russia, Siberia, Norway, Sweden, Turkey, and Japan.

On November 1, 1939, the Montana state fish and game department submitted a dead beaver to the Montana Livestock Sanitary Board laboratory for examination. It was reported that several beavers had been found dead during the past year in the particular region from which this beaver was obtained, but this was the first time a carcass had been obtained fit for laboratory examination.

Postmortem findings revealed minute gray and white pin-point lesions in the liver and spleen. (See figure 1.) The same characteristic lesions were found in the kidneys and the mesenteric lymph glands. An excessive amount of fluid was found in the peritoneal, thoracic and pericardial cavities. Hemorrhagic gastritis and catarrhal enteritis were present in the intestinal tract.

Smears made directly from the liver and spleen revealed a non-acid-fast, Gram-negative rod. Bacteriological cultures were made on blood-cystine agar of the heart blood and material from the liver and spleen. Two guinea pigs were inoculated subcutaneously at the same time with 1 cc. each of a ground up material from the spleen and liver. Heart blood was drawn and the serum was used for the agglutination test. After 48 hours of incubation no reaction was noted in any of the dilutions.

At a later date blood from a patient that had recovered from tularemia agglutinated antigen made from pure cultures obtained from the spleen of this beaver.

A grayish-white viscous growth was obtained on the cultures after 48 hours of incubation. Smears stained with Gram's stain revealed a minute, Gram-negative rod which in many cases appeared coccoid. A hanging-drop preparation presented the organisms as nonmotile. An original pure culture of the spleen was used in inoculating various mediums. No growth was obtained in litmus milk, beef-infusion broth or nutrient broth. Slight liquefaction of the gelatin stab was noted after 24 hours of growth. A slight growth was obtained on egg-albumen agar. Slight acidity but no gas was noted in mannite, dextrose and glucose.

The diagnosis was tularemia, due probably to water-borne infection.

Tularemia has been reported by Bishop in the muskrat but this is the first time, to our knowledge, that it has been reported in the beaver.

It would appear that the beaver had been

*From the laboratories of the Montana Livestock Sanitary Board.

living in stagnant water and that the beaver population was considerably larger than it should have been in the area where the disease appeared.

We may state that the microscopic postmortem lesions were very similar to Sylvanic plague, so much so that we immediately forwarded tissues by air express to the Plague Laboratory at San Francisco, Calif. The diagnosis of the Plague Laboratory was negative for Sylvanic plague.

Oxytenia Acerosa—a Plant Poisonous for Live Stock*

IN PAST YEARS in certain canyons of western Colorado, cattle have died after eating *Oxytenia acerosa*. The poisonous nature of the plant has been demonstrated experimentally by feeding it to lambs and rabbits.

The dried ground plant (moistened with water) mixed with chicken-laying mash was fed to two rabbits, following which death occurred, both animals showing similar pathological changes.

Three lambs were force fed (stomach tube) measured amounts of *O. acerosa* mixed with water. Death occurred in six to twelve hours. A fourth lamb was force fed 244 Gm. (8 oz.) of the dried stems devoid of leaves. Death followed in about 22 hours.

In order to obtain some information regarding the poisonous principle of the plant, 1.5 Gm. (23 gr.) of tannic acid was mixed with 210 Gm. (7 oz.) of the finely ground dried plant material. The mixture was well moistened with water and allowed to remain in the refrigerator overnight. A lamb was force fed this mixture; death followed in about 23 hours.

The gross and microscopic pathology exhibited by lambs and rabbits which consumed the plant were quite constant. The gross lesions were characterized by a distention of the abdominal blood vessels, petechial hemorrhages in the mucosa of the abomasum and small intestine, and engorgement of the liver with blood. Microscopically the liver capillaries and sinusoids were distended, some of the hepatic cells showing cloudy swelling and fatty degeneration. Gastric pathology was not observed in the rabbits.

*From the Colorado Experiment Station, Fort Collins, Colo. By Frank Thorp, Jr., D.V.M., M.Sc., Ph.D.; G. S. Harshfield, D.V.M., M.S.; L. W. Durrell, B.S., M.S., Ph.D.; and C. Guinn Barr, B.S, MS., Ph.D.

Hog Ringing

By District Veterinary F. V. HOLMBOE

Stavanger, Norway

I ENJOYED reading the article by John B. Bryant, entitled "Swine Rooting and Hog Ringing," in the October 1939 issue, page 508. The readers of the JOURNAL may be interested to learn that in Norway such a ringing of hogs is forbidden by law.

In our country we have the Dyrevernloven (= the law for the protection of animals) of June 7, 1935. Therein, one is forbidden to put a string or a ring in the muzzle of pigs or make some other operation which hurts the muzzle of the pigs, with the object of restraining them from raking up the earth. In some places it was the custom to cut off a piece of the disk of the muzzle in order to keep the animals from rooting.

When the law was discussed, there was considerable criticism and debate over this provision, as in some districts this practice was not considered an act of cruelty. It was held that when the pigs are ringed, they can go on the grazing ground and enjoy a freer and better existence than when they are penned up. It was also held that by ringing the pigs the grazing grounds are better utilized and that this is of great economic importance to agriculture. It was further argued that many people have ringed their pigs for years without injuring them. The argument that our ladies wear rings in their ears also was presented.

It goes without saying that the technical execution of the operation and the material used for the ringing are of importance from a humanitarian point of view. Executed awkwardly and with poor material, ringing, as pointed out by Dr. Bryant, is a

barbarous practice. The same objection can be raised against castration.

Rings made of a moderately hard alloy of copper are probably to be preferred, since such rings protect against suppuration.

That this operation should not be made by unqualified persons is evident but whether it can be defended from the veterinary and humanitarian standpoints is questionable. In this regard, the writer would be interested to learn the opinions of American veterinarians.

Chronic Pneumonia in a Cat*

By MORRIS POLLARD, D.V.M., M.Sc.

Richmond, Va.

THE LEFT lung of an aged cat was submitted to the diagnostic laboratory of the biology department, Virginia Polytechnic Institute, by Drs. Thompson and Eggert of the Roanoke Animal Hospital, Roanoke, Va., with the following history.

The animal suffered with a respiratory disturbance for three months. During that time, it became emaciated and weak due to anorexia. The respirations were dyspneic, the mucous membranes cyanotic and locomotion ataxic. In the course of the preliminary examination the cat became excited, struggled for several moments, and then died suddenly.

An autopsy was performed and the left lung, preserved in 10 per cent formalin, was submitted for histological examination. It was found to be gray upon macroscopic inspection. It was hard and fibrous on palpation and failed to float when immersed in water. The cut surface was dense, meaty, and uniformly grayish throughout. No exudate could be detected in the bronchioles.

Microscopic sections stained with Delafield's hematoxylin and eosin showed a generalized consolidation radiating for several millimeters around the bronchioles. In these areas cellular fragments, degenerating cells with pyknotic nuclei, and several hydropic cells occupied part of the alveoli. All of these components were suspended in a fibrinous exudate. The greater part of the affected alveoli, however, were occupied by typical fibroblastic cellular elements. These consisted of collagenous fibers with numerous hyperchromatic nuclei in their interstices. These fibers were contiguous with the fibrin and with the alveolar wall proximal to the bronchiole. The interalveolar septa were thickened and infiltrated with considerable connective tissue. No hyperemia was visible.

We can assume that the lesion described above represents the sequel of a delayed resolution following a catarrhal pneumonia. The condition produced may be called an organizing pneumonia. It represents either an extension of connective tissue from the adventitia of the bronchiole or a progressive differentiation of the cellular elements of the exudate, particularly the pluripotential monocyte, which is involved during the latter stages of the cytological sequence of a chronic inflammation.

*From the department of biology, section on animal pathology, Virginia Polytechnic Institute, Blacksburg, Va.

Nephritis in the Dog and Cat

Among the animals examined post mortem at the institute of animal pathology of Munich during the year (1938), 20 dogs and more than that number of cats were found to have been suffering from glomerulo-nephritis. Among the dogs 7 were acute cases, 8 subacute and 5 chronic. The percentage of acute cases was higher in the cats (14 acute and 6 subacute). Although somewhat more common among aged animals, neither age, sex nor anatomo-pathological factors materially affect the presence of that organic disease. In cats, however, glomerulo-nephritis was found to be closely related to respiratory and intestinal infections. (*Abstract, Recueil de Médecine Vétérinaire, cxv, February 1939, p. 96.*)

The common rat may be the long sought carrier of the virus of infantile paralysis, according to a recent announcement of the U. S. Public Health Service. The eastern cotton rat is susceptible to the disease.

Infectious Bulbar Paralysis (= Mad Itch)

By B. J. FINKELSTEIN, D.V.M.
Brooklyn, N. Y.

INFECTIOUS bulbar paralysis (= pseudorabies or mad itch) is important from a clinical standpoint because it must be differentiated from true rabies. It is a peculiar infectious disease that is rapid in its course after the appearance of symptoms, terminating fatally in 24 to 48 hours.

CASE 1

A Bay gelding, about 11 years old, owned by an ice and coal peddler who had had the horse for about six years, was brought to the writer on the afternoon of June 20, 1938. The horse refused food the previous evening and that morning. When hitched and taken out on his route that morning, the animal acted uneasy in the harness, shaking his skin frequently and rubbing the head against telegraph poles or trees when the wagon stopped for a delivery. However, he covered the route and was returned to his stall at about 2:00 p. m. He again refused food.

When first seen by the writer, the horse was somewhat stiff. The temperature was 103.8° F. and the pulse full and regular, but he appeared uneasy and showed an anxious expression. Tetanus was considered, and ruled out. The owner was told frankly that there was some doubt about a positive diagnosis and that it would be best to await developments for a 24-hour period.

At 7:30 the following morning, the owner telephoned the writer to call immediately. In the night, the horse had slipped his halter and was found standing in a pool of blood that morning. He would not permit close approach for careful examination. Every minute or two he would turn on his right flank, and tear large chunks of skin and muscle from it with his teeth. His entire right side was in ribbons and it appeared as if he would eviscerate himself any moment. With difficulty, the halter was replaced and the horse was led to an enclosed lot and turned loose. Again, he would not permit close approach.

The animal was destroyed by a policeman, who shot him from the other side of the fence. Since the horse was shot through the head, no postmortem examination was made to rule out the possibility of rabies.

The virus of pseudorabies may be transmitted by the bite of an infected rat. Investigation at the stable disclosed that the place was badly infested, and recommendations were made to prevent possible recurrence.

CASE 2

A male Boston Terrier, 4 years old, which had been treated by the writer on many previous occasions, was presented after having eaten a quantity of grass the previous day. The owner stated that the dog vomited persistently and refused food. She observed that the dog's bark seemed to have changed. This, together with a partially paralyzed lower jaw, suggested the possibility of rabies. Close questioning of the owner about a possible exposure to a dog bite led the writer to conclude that the dog had not been bitten at any time. In fact, the owner could recall no instance when her dog was near enough to another to be bitten.

The temperature was 102.6° F. Indigestion was suspected. Colonic irrigation was employed and a mass of feces mixed with a quantity of grass was removed. The owner refused to hospitalize the dog. She was told of the possibility of rabies and urged to return the animal promptly if any change were observed.

The following morning the dog was returned in a much worse condition. The lower jaw was completely paralyzed and both corneas were completely covered with a thick, hardened crust that could not be removed either with warm solutions or ointment. The dog definitely resented our handling him. A tentative diagnosis of rabies was made, and the owner was advised that it would be best for the health

authorities to make further examinations. The animal was placed in a metal kennel, whereupon he immediately began to tear at himself. Even with a paralyzed lower jaw, the dog had himself in shreds in a very short time. He died at the health department shelter in a few hours.

No Negri bodies were found on pathological examination and inoculation of experimental animals with brain tissue did not produce symptoms of rabies.

Complicating Fibroid Tumor in a Dog

By W. C. GLENNEY, D.V.M.
Elgin, Ill.

THE SUBJECT was a 4½-year-old English Bull, female.

History.—On June 2, 1936, a hysterectomy was performed. Intermittent vaginal discharge developed one year later from the previous infection and responded to vaginal flushes.

8-21-39.—The dog was entered with a temperature of 105° F., anorexia, general listlessness, and frequent urination. Radiography and urine analysis were negative. The case was not diagnostic until soreness in the right paralumbar region disclosed a deep-seated abscess. Drainage was instituted, with the fistulous tract reaching nearly to the peritoneal cavity. An uneventful recovery followed and the case was discharged on September 10, 1939.

10-21-39.—The case was reëntered with the previous symptoms. Embrocation packs lowered the temperature to normal in two days and an exploratory laparotomy was performed to determine the cause of the abscess. Morphine, atropine and nembutal were given. The fistulous tract was traced into the abdominal cavity in the region of the right kidney and a fibrous growth was observed surrounding the ligated stump of the right ovary. Hypertrophy of the right kidney was evident and a mass of adhesions was found surrounding the kidney tumor and intestine. An overdose of nembutal was given to effect euthanasia.

Incision of the affected kidney revealed a chronic hypertrophy, enlarged medulla and various-sized calculi. Incision of the tumorous growth showed a nucleus of unabsorbed suture linen and a fistula leading to the external opening in the flank.

The postmortem findings and the history agree on the following diagnosis. The urinary calculi account for the frequent urination and the unabsorbed linen formed a chronic irritation that initiated the formation of the fibroid tumor. The deep-seated, obscure abscess was fed by the fistulous tract from the region of the tumor.

No doubt every small animal practitioner has used a linen ligature for strength when ligating during an oöphorectomy or hysterectomy with no questionable results. However, this case clearly demonstrates the inadvisability of using nonabsorbable suture material in abdominal surgery. Such cases do not appear every day but they are embarrassing when they do.

Rabies in Kansas City

Speaking before the Nebraska Veterinary Medical Association at the Fontenelle Hotel, Omaha, December 12, A. Trickett, general practitioner of Kansas City, Mo., reviewed the results of ten years of compulsory vaccination of dogs against rabies. The campaign, which grew out of a high incidence of rabies in Kansas City, was supervised by the local department of health and was conducted in such a way as to avoid any hardship upon owners of dogs who could not afford to pay the usual charge. The result was that rabies has been practically eliminated from that city.

The report, said Dr. Trickett, is open to the closest investigation of the opponents to single-dose vaccination as one of the measures required to control rabies in a large city.

The reduction of rabies to a negligible degree in Kansas City and similar results achieved at the army posts of this country are two examples of the value of vaccination of dogs against rabies.

Botulism and Encephalomyelitis in Horses

By FRANK HARE, D.V.M., M.S.
Lexington, Ky.

MANY VETERINARIANS believe that symptoms alone are insufficient ground for the differential diagnosis of botulism and encephalomyelitis; that, therefore, an accurate diagnosis can be made only in the laboratory. The writer is not inclined to think with the exponents of this theory.

A specific disease is manifested by a specific symptom complex, and the successful treatment of such a disease rests upon the proper interpretation of its symptoms. Many practitioners are prone to hedge the responsibilities of diagnosis *via* laboratory workers and, unfortunately, these men are usually overanxious to accept them. Laboratory findings are valuable supplements to the clinical symptoms and postmortem findings but, for the final authority on a diagnosis of encephalomyelitis, this writer prefers the consensus of his practicing colleagues. The reason: The encephalomyelitis virus can rarely be isolated from material obtained from cases that have died and it can not be recovered in every instance, even in induced cases, from the brain tissue of fresh material.

No uncomplicated case of botulism that did not run a subnormal temperature has been observed by the writer. An outbreak in Delaware is recalled wherein ten or eleven horses in a stable of 18 were stricken in one day. In every instance the temperature was subnormal; 97.2° F. was recorded for some of the cases and all were below 100. From the time the first symptoms were manifested and throughout the attack, all of the cases were observed carefully. Without exception the animals exhibited a progressive motor paralysis, but with no disturbance of consciousness. None showed a reddish yellow discoloration of the conjunctiva and, on postmortem examination, when the spinal canal was entered through the atlo-axoid space, there was not the gushing of spinal fluid found in encephalitis. After being unable to rise for several days, a few developed hypostatic or mechanical pneumonia accompanied with a rise in temperature. A subnormal temperature, however, was always present in the initial stages.

It is interesting to contrast the above outbreak with a similar disease that occurred along the eastern seaboard. The condition was manifested in the initial stages by dullness and languor but, in every instance, there was a fever, a marked disturbance of consciousness, and a reddish yellow conjunctiva. On postmortem examination, these cases showed an increase in intraspinal pressure. In spite of the fact that equine encephalomyelitis was not known to be present in the United States at the time of the outbreak, Borna disease was suspected by those who investigated the cases.

Complications, of course, confuse the clinical picture as well as the laboratory findings, but in the first stages a clinician should experience no difficulty in distinguishing between botulism and encephalomyelitis. The investigations of recent years tend to strengthen the opinion that these entities can be differentiated more accurately by the clinical symptoms and postmortem findings than by laboratory examination. There are, however, some cases that present more difficult problems than others.

An example of the above type was a grade draft horse on a farm located near one of the country's leading Thoroughbred-breeding establishments. Stationed in a shed adjoining the barn, the owner and several hired men observed the actions of the animal. According to their report, the horse walked in a circle in the pasture field. Led into the barn, the animal began to walk again and, when attempts were made to stop him, he walked through the side of the barn, taking a panel with him, and continued to walk as all hands tried to pull him

back. Finally, he bumped into a wire fence and toppled over it head first.

He was apparently not hurt and was returned to the shed. He would stand for a time and, following, would begin to walk in a circle to the left, dragging the toes and staggering. When standing, he struck unnatural positions. The lips were pendulant; temperature, 102° F.; pulse slow, full and strong; respiration normal; conjunctiva pale and yellow; and peristalsis somewhat suspended. The animal did not seem to recognize his surroundings. On passing the stomach tube through the nostril, there was some difficulty in swallowing. He was given a quart of mineral oil, a quart of linseed oil, an ounce of a cresol compound and 2 ounces of ether. At the time of passing the stomach tube the animal had an uncontrollable desire to walk; therefore, we were forced to walk by his side, steer him away from objects and guide him in a circle to the left.

He was given calcium gluconate and dextrose intravenously. The owner was instructed to keep the animal comfortable and guard him from injury. The next morning, the horse apparently had made a complete recovery.

We have treated a number of similar cases. All exhibit a pale yellow discoloration of the conjunctiva, in contrast to the reddish yellow discoloration in equine encephalomyelitis; also, the initial temperature is not quite as high. In all other respects, they present symptoms practically identical with equine encephalomyelitis.

A few years ago we visited a horse that had been showing virtually all of the brain symptoms found in encephalomyelitis. The animal was down and unable to rise, and we observed a marked disturbance of consciousness. The animal made walking movements and appeared to be moribund. No treatment was attempted and we instructed the owner to notify us when the animal died. This it did, later in the day.

On decapitating the animal, an increase in spinal fluid was noted. The head was taken to the University of Kentucky, where an area of necrosis about the size of a hen's egg was found in the cerebrum. The case was so far advanced that the symptoms and pathology were complicated. (Robert Graham of the University of Illinois has entitled this particular disease toxic encephalitis.) The animal had shown symptoms sufficiently serious to alarm the owner only for a period of ten or twelve hours. It was amazing to the writer to see the amount of necrosis in the brain, and it seems probable that such cases might present a clinical picture that could be confused with encephalomyelitis of virus origin.

Gerhard Domagk

Gerhard Domagk, of prontosil fame, was awarded the Nobel prize for medicine. He was born in 1895 and until recent years was best known in the field of pathology. After teaching that subject in two German universities until 1927, he became director of a dye works in Elberfeld, where in 1932 he demonstrated the curative action of prontosil in streptococcic infections of mice. The announcement of the discovery in medical publications was made in 1935. For his work he was awarded the Emil Fischer medal of the German Chemical Society in 1937 and the Cameron prize of the University of Edinburgh in 1939.

The orthodox treatments of sterility in cows and mares should be considered only as a repair of a link in the chain of the reproductive process without strengthening the chain itself. The fact that a cow becomes pregnant after treatment does not prove that she has been made a profitable breeder. Such a cow, according to Williams (1939), is generally an inferior breeder thereafter.

Stilboestrol, the new synthetic female sex hormone, is a coal-tar derivative. It is said to be six times stronger than the natural hormones it represents, has the added advantage that it can be taken by the mouth, and is much less costly. It was discovered by Professor E. C. Dodd of Oxford (England), who showed the altruistic spirit in refusing to take out a patent.

The Occurrence of Erysipelas in Turkeys

By CARL F. SCHLOTTHAUER,* D.V.M., and LUTHER THOMPSON,† Ph.D.
Rochester, Minn.

AMONG lower animals erysipelas occurs most frequently in swine, but according to Hutyra, Marek and Manninger[1] it has been observed in nearly all species of domesticated animals and fowl. The importance of its occurrence in birds is not appreciated by all persons. It has been observed to cause severe losses in turkeys. Outbreaks of erysipelas in turkeys were reported by Beaudette and Hudson[2] in 1936 and by Madsen[3] in 1937. Because of the economic importance of this disease in turkeys, its occurrence in another flock is herein reported.

On November 21, 1938, two turkeys were brought to us for examination and diagnosis. One turkey (no. 1) was dead and the other (no. 2) was alive but moribund. They were secured from a flock of approximately 100 turkeys 6 to 7 months of age. The owner stated that this flock had been in apparent good health until November 19. Since then, several turkeys had died and many were sick.

The symptoms manifested by the sick turkeys were not diagnostic of any one disease. The birds presented a dejected appearance; the head was lowered, the wings drooped and their feathers were ruffled. The wattles of those more critically ill were dark and cyanotic. The fecal discharges from some of the sick birds were semifluid and yellow or green.

Necropsy revealed hemorrhages in the muscles of the thighs and abdomen. Numerous petechiae were present on and in the heart and intestine. The liver and spleen were markedly enlarged and friable. Many necrotic foci or infarcts were present in the liver and spleen from turkey no. 1, causing these organs to have a mottled appearance. The kidneys were slightly enlarged and congested. Similar lesions were observed in other turkeys from this flock that died during the course of this disease. Thirty to 40 per cent of the entire flock succumbed.

A pure culture of a small, slender, Gram-positive rod was obtained from the liver and heart blood from each turkey. This organism had the following cultural characteristics.

Growth on all mediums used was scanty. The addition of blood made little difference in the amount of growth. Dextrose brain broth developed a slight turbidity and an acid reaction. Nutrient broth was slightly turbid in 24 to 48 hours but became clear after seven days. On nutrient agar a slight, transparent growth appeared after 24 hours. A hand lens was used in order that we could determine whether the slight roughness of the surface was actually due to colonies of the bacteria. On all of these mediums the organisms developed as thin, Gram-positive rods of varying lengths, with an occasional short filament. Blood-agar-poured plates showed tiny, deep colonies after 48 hours surrounded by a small zone of partly hemolyzed red cells. The most characteristic growth occurred in gelatin stab cultures, incubated at room temperature. Growth spread laterally from the stab to form, in about seven days, the typical test-tube brush appearance which is characteristic of *Erysipelothrix rhusiopathiae*. There was no liquefaction of the gelatin.

On November 22, two rabbits were each inoculated intravenously with 2 cc. (30 minims) of a 24-hour-old primary pooled glucose broth culture obtained from the heart blood and liver of both turkeys. Both rabbits died on November 24. A small, slender, Gram-positive rod, morphologically and culturally identical with that obtained from the turkeys, was obtained from the heart blood of each rabbit.

On November 25, a third rabbit and two

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white leghorn chickens 2 to 3 months of age were inoculated with 24-hour-old broth cultures obtained from the first two rabbits. The rabbit received 2 cc. of this culture intramuscularly; the chickens each received 0.5 cc. (7 minims) intravenously.

The rabbit died on November 27 but both chickens survived and remained in apparent good health.

The source of the infection in this flock of turkeys was not determined. These turkeys were hatched in an incubator and reared in a brooder house and an outdoor pen that did not permit contact with the ground. The brooder house has a solid board floor and the outdoor pen has a coarse wiremesh screen floor 2 feet or more above the ground at its lowest level. The turkeys never came in direct contact with other fowl or domesticated animals. They were fed a commercially prepared turkey food, which was placed in a trough hanging on the outside of the pen. The owner stated that he entered the pen only when it was necessary to do so to remove feathers and droppings from the screen.

This farm maintained a small herd of swine. The young pigs frequently escaped from their pen and were often observed walking about in the droppings underneath the turkey pen. Yet, none of these swine contracted erysipelas. During the latter part of October two other herds of swine situated within 1 mile of this farm suffered severe losses from a malady that was later diagnosed as swine erysipelas. A large flock of turkeys was maintained on one of these farms and there was ample opportunity for infection to be carried from the infected swine to the turkeys, but the latter remained healthy. It seems unlikely that the infective organism that we isolated from the turkeys was identical in pathogenicity with that causing erysipelas in the two herds of swine 1 mile distant.

References

[1]Hutyra, F., Marek, J., and Manninger, R.: Special Pathology and Therapeutics of the Diseases of Domestic Animals. 4th English ed. (Alexander Eger, Chicago, 1938) i, pp. 76-95.

[2]Beaudette, F. R., and Hudson, C. B.: An outbreak of acute swine erysipelas infection in turkeys. Jour. A.V.M.A., lxxxviii (1936), n. s. 41 (4), pp. 475-487.

[3]Madsen, D. E.: An erysipelas outbreak in turkeys. Jour. A.V.M.A., xci (1937), n. s. 44 (2), pp. 206-208.

Chicken-Pox Vaccination

In replying to the inquiry of a member practicing in the tropics relative to the virtue of pigeon-virus vaccine as compared with the chicken-pox type, which has caused considerable controversy between European and American poultry pathologists, we stated: "Although no one questions the gentle action of pigeon-virus vaccine as compared with the chicken-virus type, American poultry pathologists are in accord as to the superior protective properties of the latter. The consense among them is that pigeon-virus vaccination does not confer a degree of immunity sufficient to justify its exclusive use in controlling chicken pox in American flocks."

Carotene for Layers

Williams, Lampman and Bolin (*Poultry Science*, July 1939) set at 0.2 mg. per bird daily the amount of carotene required to maintain weight, fair egg production and hatchability. This amount also wards off deficiency-disease lesions of the throat.

The source of the carotene was alfalfa meal properly assayed and stored to control vitamin A depletion. Two mg. per bird daily is obviously close to the minimum amount of carotene laying hens require to lay well and thrive, since 0.1 mg. per bird daily was found to be definitely insufficient. The figure corroborates the work of Sherwood and Fraps (1934), who set at 240 Sherman-Munsell units the amount of vitamin A laying hens require to keep in good health. (Two mg. of carotene is equal to 238 S-M units, or 333 U. S. P. units of vitamin A.) Larger amounts of carotene per day are, however, required to increase the vitamin A content of the eggs and insure good livability of the chicks hatched therefrom.

The average daily purchase of merchandise from the United States by Japan is about $700,000.

Black Disease Immunization*

By E. A. TUNNICLIFF†, D.V.M., M.S.

Bozeman, Mont.

IN A PREVIOUS paper[1] there was reported the preparation of an alum-precipitated toxoid capable of immunizing sheep against infectious necrotic hepatitis. Experimentally vaccinated sheep were immune to at least 34 mld of the toxin when injected intravenously 251 days after vaccination. The remaining unexposed sheep in this vaccinated group were held to determine the duration of immunity. However, some unrelated deaths reduced the number of animals in this group, and it seemed advisable to expose the survivors at 17 months rather than to wait longer, as originally planned.

These sheep had been vaccinated by the single injection method, which is the recommended procedure in this country under field conditions. There were also available a number of sheep that had been twice vaccinated 17 months previously, and these were included in the test. Vaccination tests to determine the full duration of immunity are under way at this laboratory, but since the final results will not be available for some time, an account of the results to date seems appropriate.

EXPERIMENTAL PROCEDURE

The animals used for this test were all mature ewes in good physical condition, varying in weight from 103 to 158 pounds. The flock from which they were procured was the same as that reported in the previous writing.

The immunized sheep were injected subcutaneously with an alum-precipitated toxoid prepared from *Clostridium novyi (edematiens)* isolated from sheep dying of infectious necrotic hepatitis. Two animals, numbers 1 and 2, received one 5-cc. dose on May 14, 1937, and the other seven, numbers 3-9, received two doses on May 14 and 22, 1937.

Preliminary titrations to determine the mld of toxin were made on September 29 and 30, 1938. Sheep 10 survived the intravenous injection of 0.3 mg. of precipitated

*From the Montana State College, Agricultural Experiment Station, paper No. 129, journal series. Montana Experiment Station and Montana Livestock Sanitary Board coöperating.

†Montana Veterinary Research Laboratory.

TABLE I—Immunity test of vaccinated sheep.

Sheep Number	Date Vaccinated* (1937)	Exposure†				Result
		Date 1938	Material	Dose		
				Mg. per Pound of Body Weight	Mld	
1	5–14	10– 6	Precipitated *C. novyi* toxin	12.6	34	Lived
2		10–10		12.6	34	Lived
3	5–14 and 5–22	10– 4		12.6	34	Lived
4		10– 6		12.6	34	Died
5		10– 6		12.6	34	Lived
6		10– 6		12.6	34	Lived
7		10– 6		12.6	34	Lived
8		10–10		12.6	34	Lived
9		10–10		12.6	34	Lived
10	Controls	9–29		.30	1	Lived
11		9–29		.37	1	Died
12		9–30		.37	1	Died
13		10– 6		.37	1	Died

*Dose of alum-precipitated toxoid, 5 cc. subcutaneously.
†Intravenous injection.

toxin for each pound of body weight, while sheep 11 and 12 died when injected with 0.37 mg. per pound, which was accepted as the mld.

The nine vaccinated sheep were tested for immunity on October 4-10, 1938, 17 months after vaccination, by the intravenous injection of 34 mld (12.6 mg. per pound of weight) of precipitated toxin. An additional control sheep, number 13, included with the test on October 6, received 0.37 mg. or one mld of toxin. This control and one of the vaccinated sheep, number 4, died, but the other eight vaccinated sheep were solidly immune.

CONCLUSION

The single vaccination with a 5-cc. dose of alum-precipitated toxoid immunizes sheep against *Clostridium novyi* toxin for at least 17 months.

Reference

[1]Tunnicliff, E. A., and Marsh, H.: An alum-precipitated toxoid as an immunizing agent against infectious necrotic hepatitis (black disease) in sheep. Jour. A.V.M.A., xciv (1939), n. s. 47 (2), p. 98.

Avian Tuberculosis

An enlightened husbandry should not tolerate avian tuberculosis. The disease causes manifest unthriftiness and lowered egg production and, besides being transmitted to swine and sheep, it causes confusion in testing cattle by sensitizing them to tuberculin. Although the disease seems to be increasing, no definite plan of control has been instituted.

The disease is most prevalent in the North Temperate Zone. In the North Central States 50 per cent of the chicken flocks are affected. In affected flocks the percentage of fowl tuberculosis ranges from 5 to 95 per cent. For the whole United States the incidence appears to be 5 to 6 per cent of all chickens. In one survey comprising 28 infected farm flocks, the occurrence was 11.1 per cent of the 1,476 adult chickens tested. The incidence among the pullets (under 1 year of age) of the same flocks was 0.19 per cent.

The failure to reduce the percentage of swine tuberculosis by eradicating the disease in cattle shows that avian tuberculosis is responsible. Unhygienic environment is the cause. There is no convincing evidence that eggs laid by tuberculous hens are a factor. Infected fowl may live for years or the infection may be acute and cause death in a few months. The disease is afebrile. Lameness, loss of weight and general unthriftiness in spite of a good appetite, and the chronicity are revealing signs but convincing proof lies in the post-mortem findings. The intradermal test preconized by Van Es and Schalk (1914) is helpful but not infallible. Infected fowl may not react and *vice versa*. Familiarity with the characteristic organic lesions is essential. The lesions are teeming with the specific organism and since ulcerations in the intestine are common, many are strewn about with the droppings.

Control of avian tuberculosis is not a simple matter, since the avian tubercle bacillus may remain viable in soil for years. The Freidlander plan of vaccination, based upon the principles of BCG, is not justified in view of the results obtainable. The disposal of all fowl after each laying season has merit but that the disease can be thus eradicated remains to be proved. *(William H. Feldman. Veterinary Medicine, xxxiv, December 1939, pp. 734-742.)*

In given species of domestic animals, the functioning of the genital apparatus of the female differs to a certain extent with breeds in regard to the time and intensity of estrus and ovulation, but it always follows the same sequence of events. To obtain fecundation with the greatest degree of certainty, it is, therefore, indispensable to know the propitious moment for copulation.—*Berthelon.*

Though piroplasmosis and babsiellosis respond to trypanblue, gonacrine and acaprine, there is no specific medication for anaplasmosis and theilerosis. — *Ch. Alla. Thèse, Lyon, 1937.*

SURGERY & OBSTETRICS

Surgical Technics in Glaucoma*

By MASON WEADON, V.M.D.
Washington, D. C.

BEFORE entering into a discussion of the operative technics for the relief of glaucoma, it seems necessary for one to review the anatomy of the eyeball with special reference to the exact boundaries of the cavities known as the anterior and posterior chambers of the eye.

The anterior chamber of the eye is that part which is anterior to the iris and the anterior surface of the crystalline lens. Of course, this chamber is bordered anteriorly by the cornea. The anterior chamber is many times larger than the posterior chamber, which is a small, triangular surface that on a cross section of the eye from top to bottom through the middle would appear bordered anteriorly by the posterior surface of the iris, a small part of the anterior surface of the lens which forms the base of the triangle and posteriorly by the ciliary processes.

As this is rather an involved explanation, the writer wishes to point out that both the anterior and posterior chambers of the eye are anterior to the lens and the capsule of the lens.

An understanding of these anatomical details will help one to overcome the popular impression that the anterior chamber is all that part of the eye which is anterior to the lens and that the posterior chamber is that part of the eye posterior to the lens which is known as the vitreous body. Thus, the vitreous body is not the posterior chamber of the eye.

The cause of glaucoma is not definitely known, but the adage that it is "dropsy" of the eyeball seems to explain the condition as we find it. In other words, glaucoma is an accumulation of excess lymph-like fluid anterior to the lens. The normal flow of the lymph is from the posterior chamber into the anterior chamber into the canals of Schlemm, which are located at the junction of the iris and the cornea and the sclera. All of these surfaces in the anterior and posterior chambers of the eye, mentioned above, are nonabsorbable tissues. Therefore, the function of the canals of Schlemm is to conduct the excess lymph to the sclera or conjunctiva, which are absorbable membranes—hence, to regulate the pressure of the fluids of the eye, by draining off the excess, by conducting these fluids to absorbable membranes or lymph channels of these tissues.

When the canals of Schlemm become clogged or do not perform their function, there is a collection of fluids in the anterior and posterior chambers of the eye, with an increase in intraocular pressure. This is the condition known as glaucoma. Why these ducts become clogged, no one can say positively but the writer's theory is that this is a hereditary tendency. This belief is founded upon the fact that all of the offspring of a stud dog in the writer's locale have developed glaucoma, if they have lived to a mature age.

At this point it might be well to inject a practical method for use in everyday practice for the diagnosis of glaucoma. Outside of a highly technical examination by a trained ophthalmologist, there are three points which, if established, will confirm a diagnosis of glaucoma.

First, the normal consistency of the eye-

*Presented at the small animal clinic of the 76th annual meeting of the A.V.M.A., Memphis, Tenn., August 28 to September 1, 1939.

ball, under pressure from the finger, is similar to that of the ordinary worm capsules, while in a glaucoma the pressure is so increased that the eye feels as hard as an apple. Second, the iris is widely dilated and pushed back into convoluting folds. Third, the eye is much larger than normal, sometimes approaching the size of a golf or billiard ball, depending upon the size of the dog. The cornea, due to the irritation and injection of excessive lymph, is blue.

For the relief of this condition there are four operations. First, with a scleratone (= a triangular spade-like instrument, sharp on two edges, with a point) an opening is made through the conjunctiva and sclera about one-eighth inch back from the cornea into the anterior chamber of the eye. Through this opening the iridectomy forceps are passed, the iris is grasped and pulled back through this opening and a triangular area as large as the pressure on the iris will permit without tearing is snipped off and the remnant is allowed to slide back into position. Through this opening in the iris the fluids pass back and forth between the two chambers more freely. In cases diagnosed very early this operation is helpful. The lids are stitched together in this operation, and subsequent operations for a period of six to ten days.

Second, the procedure is the same as the first with the exception that when the iris is pulled through the wound, instead of its being cut off it is cut into two flaps which are pushed, one to the right and one to the left, between the conjunctiva and the sclera. In many cases these two flaps of the iris which have been pulled through the incision will stay in position between the sclera and the conjunctiva of their own volition; however, in closing the wound with four O catgut, it is well to include the free edge of the iris and thus fix the iris in place. In this way we create artificial canals of Schlemm; that is, we now have an opening through which the fluids can readily communicate with an absorbable membrane.

Third, in all chronic cases where the eye is greatly increased in size, the cornea very blue and the pressure has reached almost the bursting point, the writer recommends as an almost certain cure the removal of the lens in its entirety. This is done by making an incision about one-eighth inch back from the cornea through the conjunctiva and sclera into the anterior chamber of the eye. Through this incision the iridectomy forceps are inserted so as to grasp the capsule of the lens, which is soft and has the consistency of jello and is not organized and hardened as in cataracts. Therefore, it is necessary to put pressure on the lower edge of the lens and its capsule by means of a round, ordinary operating hook at the same time that the capsule is grasped and pulled through the opening. In other words, one pulls with the forceps and pushes with the hook, which is applied at the volar surface of the lens through pressure exerted on the cornea. It is well to cover the cornea with sterile mineral oil and dip the round hook in mineral oil in order to reduce the traumatism of the cornea to the minimum.

Fourth, a certain cure for the relief of glaucoma is the removal of the eyeball. This may sound somewhat absurd but in those cases wherein only one eye is involved and the other eye is apparently normal, Dr. Cashell and the writer have found that if the affected eye is removed, in a large percentage of cases the other eye will not become affected.

In all of these operations the highest possible antiseptic precautions must of course be observed. All instruments are boiled and immersed in alcohol. The hair and the lashes are shaved away and the eye is washed with six to twelve eyedroppers full of 1 per cent solution of acriviolet. It is sometimes helpful, after the animal is asleep, to desensitize the eye by the application of a 1 per cent butyn solution because the eye retains its mobility in many cases under the deepest anesthesia which one may give. In this connection it might be added that in these operations the usual nembutal anesthesia is used.

In some cases where the intraocular pressure is great, the use of 1 per cent eserine salicylate in an ointment base applied to the cornea lessens the intraocular tension.

The application of this treatment every four hours for 24 to 48 hours before operating is very helpful, and also in cases where the owner will not allow an operation. This ointment contracts the iris and relieves the pressure as long as it is used. However, the continuous use of this will irritate the mucous membrane and the cornea of the eye.

Oxalic Acid as a Hemostatic

Though the mechanism of its dynamics is not known, oxalic acid solution is said to possess unusual value as a hemostatic agent. The action is a paradox, since oxalic acid prevents coagulation *in vitro*, and does so by precipitating calcium (Sollmann). Notwithstanding this effect, intravenous injections of a 5 per cent solution in safe dosage are said to stop bleeding. The safe dose of a 5 per cent solution and does so by precipitating calcium (Sollarge animals (horses and cattle) 100 cc. (3.3 oz.) is prescribed. Since oxalic acid is a deadly, corrosive poison, it should be used medicinally only with considerable care.

Red Cell Concentration in the Diagnosis of Shock

Shock is a reduction in the blood volume either through loss or pooling. The pooling in animals, because of their extraordinarily vast splanchnic circulation, generally takes place in the ramifications of the mesenteric arteries, which in a state of relaxation are capable of receiving a large enough proportion of the total blood volume to deprive the periphery of vital irrigation.

In secondary shock, the pooling may be in the form of plasma that has escaped through permeable capillary walls into the tissues. To what extent this type of shock occurs in animals subjected to major surgical operations has not been studied. Shock has been overlooked to a considerable extent in animal surgery. Secondary shock is seldom mentioned in the literature. It has been more convenient to attribute delayed postoperative deaths to infection.

According to theories originated during the World War, secondary shock is caused by toxic material, absorbed from the wound, that weakens the capillary walls to such a degree that large amounts of plasma transude into the perivascular spaces and leave the blood remaining within the vessels in a state of high cellular concentration. The count of red cells is high and may therefore be used as a diagnostic measure.

In short, a marked increase of erythrocytes in a patient becoming progressively weaker is an evil sign following a surgical operation.

Blood Banks

The use of blood banks, or blood stored for therapeutic use, dates back to 1916. The practice, which was widely publicized by the institutes of hematology at Moscow and Leningrad, is of World War origin. Rous and Turner (*British Medical Journal*, June 22, 1916) demonstrated that blood values of rabbits depleted of large volumes of blood could be restored by the transfusion of equal amounts of homologous blood preserved in a citrated solution for two weeks. It was, however, the Russian hematologists who proved the feasibility of the practice in everyday clinical work (human).

Since then, Ascoli and Vercesi of Italy (1934) introduced the use of placental blood for therapeutic transfusions in lieu of whole adult blood. The advantage of placental blood lies not only in its inexhaustible supply but also in its stimulating properties and high content of hemoglobin, red blood cells, and bilirubin. It stimulates the blood-forming mechanism and thus overcomes anemia and hemorrhagic diatheses.

The technics of collecting and preserving placental blood are now standardized in human medicine. To what extent the new scheme may be found utilitarian in animals remains for the future to decide.

Although statistics are entirely lacking on the mortality from surgical operations in animals, there is no doubt that the figures are high—too high for this age of improved diagnostics and sterilization.

That Troublesome Third Maxillary Molar

To the Editor: A Percheron mare, 3 years old, weighing 591 kg. (1,300 lbs.) has a decayed third maxillary molar that can not be extracted with forceps. Knowing the difficulty of removing it by trephining and punching, I would like to be advised as to what should be done to remove it.—W. F. H., Oklahoma.

THE THIRD maxillary molar at the age of 3 is a troublesome unit of the dental mechanism, as the tooth often arrives at the date of eruption in the state of structural deficiency and prey disease. Its deciduous predecessor may not shed promptly because the decayed permanent is not capable of undergoing the necessary longitudinal expansion that forces out the deciduous one. Often, this interruption of the shedding sequence is manifested by quite a large bulging of the skull. The bulging itself is not a signal for immediate intervention. The tendency is toward recovery unaided. When the molar decays, however, it must be removed to prevent years of suffering and interference with normal mastication which, in the equine species, is never a trivial matter.

The extraction of a decayed third maxillary molar is best accomplished by removing the entire external alveolar plate from root to gums and prying it loose with a strong lever. A strong wood chisel must answer the purpose because there is no special instrument made for that purpose. The alveolar plate is removed either with the bone saw or common chisel.

The anesthesia consists of a procaine infiltration of the parts invaded and also an injection into the infraorbital canal.

New Method of Detecting Trouble in Anesthesia

The previous state of the patient, the surgeon's bombardment, the blood loss, and the toxic action of the anesthetic combined contribute to the cause of operating-table catastrophes. However, the anesthesia gets all the blame when death occurs before the patient is returned to its bed. This being the case, anesthetists safeguard the reputation of their art by being watchful—by detecting the first signs of approaching trouble. By this token, anesthetists of the human surgeon have been able to reduce the mortality to negligible figures, as far as the anesthesia itself is concerned. The latest development in this connection is determining the darkening of the blood at the earliest possible moment, that is to say, the earliest moment blood begins to lose its ability to carry oxygen. When the surgical patient's life is precarious, quick and early action may be necessary to prevent death.

At the 29th annual clinical congress of the American College of Surgeons, Roy D. McClure spoke of his plan of determining early changes in the blood of a profoundly anesthetized subject. A strong light, placed behind the patient's ear, makes the color changes visible by means of a sensitive photoelectric cell which registers them. Thus, the very first evidence of approaching trouble is detected.

Closing Hernial Orifices

The use of the sac wall as a pad to close the orifice in radical operations for hernia, such as is done in Macewen's operation for closing the internal inguinal ring, is a practice wide open for standard technics in animal surgery. Umbilical and traumatic ventral hernias, owing to the thick and firm walls commonly present, are examples where padding the orifice with the sac is excellent surgery, compared with sacrificing the sac and trying (in vain) to close the orifice with sutures.

"Use what you have" to close the orifice was the dictum of a onetime famous specialist in herniology. The procedure of "sacrificing what you have" is a measure too long retained in veterinary practice.

There are 120,000 pharmacists in the United States, 54,000 drug stores and 72 colleges of pharmacy.

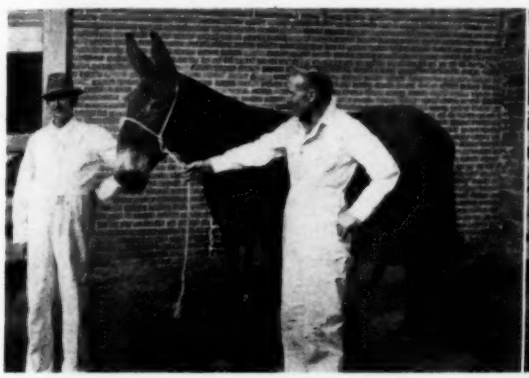
The Restraint of Solipeds in Surgery

THE SURGICAL restraint of the domestic soliped is unique. Compared with the awkward bovine and the smaller species, there are powerful, quick-acting movements and tremendous weight to be annulled by strategy—by methods of outwitting an alert animal that has been trained to yield obediently only within certain limitations and always keenly resentful to unaccustomed force.

The responsibilities assumed and the safety of all concerned are likewise unique. The patient, the doctor, and the helpers can sustain grave—even fatal—injury in every attempt to perform a surgical operation upon a horse or mule, regardless of the degree of docility. Moreover, the difficulties of positioning the site of the invasion and the terrific muscular efforts are factors which make equine surgery stand out from that of other animals.

The fact that almost all equine surgery is performed in the open, unaided by classical operating-room equipment, also taxes the surgeon's ingenuity, an ingenuity that has led to about as many methods as there are surgeons. Few veterinarians restrain their surgical patients precisely the same. In other words, this important part of equine surgery was never standardized, except to a certain extent in well-appointed surgeries where operating tables and stocks of one sort or other are standard equipment.

In view of these facts, it is not astonishing that a demonstration of unusual skill in this connection is attractive at a public clinic. Everyone, aware of the complexities of the art, is anxious to improve his methods, anxious to add new details to his favorite system. This being the case, the Memphis clinic was planned to feature this detail of large animal surgery. W. L. Stroup of Corinth, Miss., who has distinguished himself locally as a past-master of surgical restraint, was drafted to carry out a demonstration of his artful practices. While considerable publicity has been given to them pictorially in other publications since the Memphis meeting, fruitful descriptions have been lacking. As a consequence, much of the valuable instruction is apt to be overlooked or forgotten.



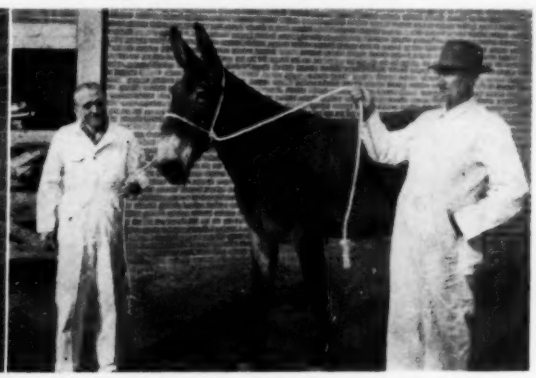


Fig. 1 (left). Draw halter. Fig. 2 (right). Dental halter.

Because of the importance of restraint in large animals it was deemed an obligation to the readers of the JOURNAL to describe in this and coming issues some of the more useful methods employed by Stroup.

Skillful restraint of large animals is not a prosaic fringe of the veterinary service. Next to diagnosis, it is the salient phase of a surgical operation and, unquestionably, the golden opportunity of distinguishing the trained from the untrained doctor. As a salvage unit of the service, surgery, moreover, ranks high. As a source of well-earned income, it has no peer in private

practice. Summed up, to be artful in the restraint of solipeds in surgery is an attainment worth while.

Figure 1 shows that the lack of an effective halter need be no deterrent. A simple length of rope, without a knot, can be maneuvered into a solid draw halter. It will fit any animal of any size (horse, mule, colt, calf, hog or dog) and can be made with a handy shank or a long snubbing rope to fill any emergency in the field. Such a halter is always an improvement over a worn-out halter, which is apt to break at an unfortunate moment.

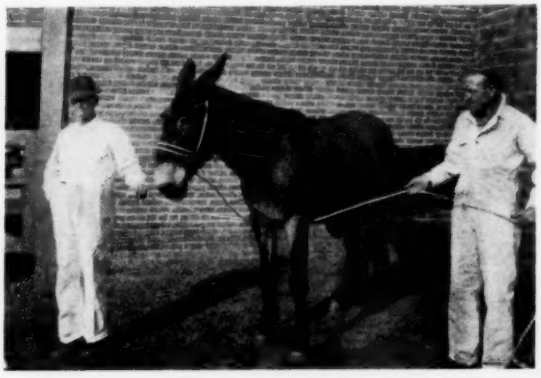


Fig. 3. Bronx halter.

The Bronx halter is shown in figure 3. This is a convenient way to make a raring horse behave. When the ropes are passed between the legs and drawn, the head is pulled downward. The subject can be made to turn a veritable summersault. Otherwise, it is a plain halter with a double shank, differing only from the Hackimore, when the ropes are passed between the fore legs. Its main use is to subdue a raring horse or as a twitch with additional control of the head.

Figure 2 is a home-made dental halter. It is similar to figure 1, except for the loops made from strands of the rope. With the two shanks the animal's head is secured by tying them to the pillars of the stall in the fashion used for the regular dental halter. The JOURNAL does not recommend this improvised halter as the equal of the standard pattern for the reason that free opening of the mouth permitted by the standard type is prevented by the part of the rope running under the mandible. For uninterrupted opening of the jaw and overcoming pressure upon the buccal walls (cheeks), which permits free access to all parts of the dentures and oral cavity, only the iron nose piece answers all purposes for which dental halters are employed. However, there is no objection to this or other rope or strap types where the mouth speculum is used.

Ovulation in the Cat

The estrual cycle of the cat differs from that of other domestic animals. It is not complete. Coitus has a provocative influence on ovulation. In examining the ovaries of cats at different hours after copulation, Courrier and Gros found that the dishiscence of the Graafian follicles occurred 26 to 27 hours after the sexual act. Ovulation in cats can also be excited mechanically—by manipulations of the vulva (Gruelich). In this manner, intense estrual manifestations and rupture of the ovisac can be brought about in approximately 25 hours after the excitation.

In classical literature the cat is said to present two estrual periods a year. In reality, according to Berthelon, the functioning of the genital organs of cats varies a great deal with breeds. Heat appears as the Graafian follicle develops. If ovulation is not provoked by copulation, the ovisacs persist for six to twelve hours and then regress. As new follicles develop, heat rapidly reappears. When ovulation is provoked, heat symptoms cease 24 to 48 hours later and a corpus luteum evolves. If not fecundated, the corpus luteum persists for one to two months and then regresses to permit other follicles and a new heat period to develop. As a rule, estrus reappears two months after parturition.

Surgical Bacteriology (1891), by Nicolas Senn, distinguished American surgeon of the 19th century, describes erysipeloid as an infective dermatitis contracted by handlers of animals and animal products, but does not, however, associate the disease with swine erysipelas.

Resection of the Vocal Cords to Suppress Barking

REMOVING the vocal cords to prevent barking, known in American veterinary literature as ventroculocordectomy or Whipple's debarking operation, was signalized for the first time at the clinic of the American Veterinary Medical Association at Portland, Ore., in 1925. The operation was performed on dogs by Fred Whipple of Peoria, Ill., and on goats by C. A. White of Los Angeles, Calif., who had worked independently and unknown to each other in creating a suitable technic.

The publicity given these operations aroused unfavorable comment from humane societies. The rationale was protested. The operation was called cruel. Whatever may be the opinion in that regard, the fact remains that an operation intended to improve the value of a domestic animal, that is humanely performed and accomplishes a laudable purpose, is justifiable. A bleating goat or a barking dog can be a nuisance to a whole neighborhood. "Should the animal be killed or improved?" are the questions to answer. By the same token, castration and spaying may be dubbed cruel.

In an article* entitled *Résection des cordes vocales chez le chien pour supprimer l'aboiement*, Le Roux and Nguyen-Dinh-Lam of the Pasteur Institute of Nhatrang found this operation important in their work on antirabic vaccination. The work required the collection of arrant dogs, many of which were inclined to bark day and night under confinement. The noise that these dogs made was intolerable to the neighborhood. In his book *A l'ombre de Pasteur*, Adrien Loir tells how the chickens, pigs, and dogs of Pasteur were a constant source of annoyance to the inhabitants of rue Vauquelin.

Rumor has it that Pasteur "performed a little operation that made dogs aphonic." The operation is not described but the presumption is that Pasteur did something to the vocal cords.

Whipple snipped out the vocal cords of profoundly anesthetized dogs with a punch forceps, a special mouth speculum and an ophthalmoscope. White invaded the larynx of goats from without.

The Nhatrang authors (cited previously) prefer the White method for dogs and describe their operative technic as follows: The dog is held in the dorsal position on a common table. One assistant holds the hind legs backward, another draws the fore limbs in a backward direction and still another holds the head and neck in extension. The laryngeal region is disinfected and anesthetized with cocaine. Three small injections of the cocaine solution insure good anesthesia.

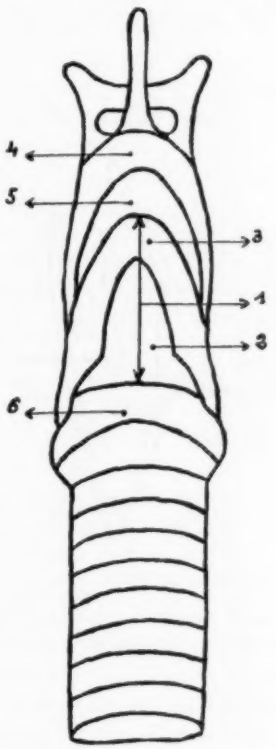


Fig. 1. (Reproduced from Recueil de Médecine Vétérinaire, March 1939.) Inferior face of the laryngeal region. 1) Incision, 2) orico-thyroidean membrane, 3) body of the thyroid (= Adam's apple), 4) body of the hyoid bone, 5) thyrohyoidean membrane, 6) cricoid.

The first step consists of an incision of

*Recueil de Médecine Vétérinaire, cxv, March 1939, pp. 154-158.

the skin and underlying muscles, 4-5 cm. (1.57-1.96 in.) long, extending from the body of the thyroid (=Adam's apple) to the level of the cricoid.

The second step is an incision from behind forward through the crico-thyroidean membrane and body of the thyroid. This is accomplished with a bistury guided forward with a cannula previously inserted through the membrane at the level of the cricoid. When the edges of the wound are drawn apart with the retractors, the vocal

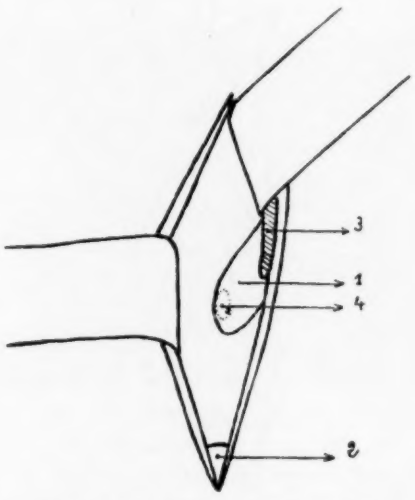


Fig. 2. (Reproduced from Recueil de Médecine Vétérinaire, March 1939.) Operative wound. 1) Left vocal cord, 2) cricoid, 3) posterior part of the body of the thyroid, 4) vocal process of the arytenoid.

cord of each side is in plain view. The anterior insertion is at the posterior third of the incised body of the thyroid and the tree border in this position is seen directed from above downward and slightly from before backward, to the vocal process of the arytenoid.

The third step is the resection of the vocal cords. The retractor on the side of the resection is slipped forward to the anterior angle of the wound. This brings the entire cord into view, from its anterior insertion to the body of the thyroid to its posterior attachment to the arytenoid. The cord is seized with a forceps and snipped out with one stroke of the scissors. The opposite cord is resected in the same manner.

The fourth step is closing the wound. The musculature is sutured with three sutures of catgut and the skin with three of silk. The advantage of disinfecting the edges of the wound with ether is pointed out. The wound and region are painted with tincture of iodine and bandaged.

Among the several hundred dogs thus treated, accidents were rare. An occasional death apparently from shock and others from hemorrhage into the bronchial tree, which caused suffocation, are mentioned as sequels. Although about 30 per cent of the dogs tore out the cutaneous sutures by scratching, the musculature always remained intact, and in no case was the larynx itself opened up. The muscles always cicatrized rapidly and well. As a rule, when examined at the end of a week to remove the sutures, the wound is almost completely healed.

In general, the results of the operation are satisfactory. Barking is suppressed or at least attenuated. If sometimes transformed into a raucous blast, the dog soon tires of making the effort.

In résumé, resection of the vocal cords of dogs is a benign operation and one easily performed. It causes either complete aphonia or satisfactory modification of the voice.

Vitamins in Surgery

Observations have shown that the administration of vitamins to surgical patients, before and after operating, greatly increases the percentages of success, according to a report read before the College of Surgeons. Many patients needing surgical operations are vitamin deficient, the report states, and the use of vitamins improves healing and builds up resistance to pneumonia and other grave postoperative accidents. Vitamins A, B, C and K (the latter to prevent hemorrhage) are the ones usually lacking in the average diet.

The word analgesia should be used to signify the abolition of pain with no loss of cerebral function.—*Bickman's Operative Surgery.*

CURRENT LITERATURE

ABSTRACTS

Lipoidosis of the Cornea of Dogs

The circular (or ovoid) and usually bilateral lesion affecting the cornea of dogs was studied by Robin and Charton (*Recueil de Médecine Vétérinaire,* June 1939) with the view of determining its nature. Although authors have described this ocular phenomenon as a keratitis (*keratitis chronique nacrée*), it is not an inflammatory lesion and it does not correspond in character to cicatricial tissue. There is neither vascularization of the sclero-corneal layers nor congestion of the conjunctiva, and neither lacrimation nor photophobia of a true keratitis. Moreover, its symmetrical and bilateral aspect distinguishes this circumscribed opacity from an otherwise damaged eyeball. It attacks adult and aged dogs, rarely subjects of 1 or 2 years of age, and the lesion is never more than 3 to 5 mm. (0.118 to 0.196 in.) in diameter, pearly and slightly rough and confined to the middle layers of the cornea.

Occasionally, the epithelium is surmounted but it always remains intact. Examined with a magnifying glass with oblique illumination, the opacity is sometimes presented in the form of irregular, concentric circles closely arranged, and in other instances its regular, neat contour gives it the appearance of a fish scale. Except in aged subjects, where other ocular affections may coexist, the trouble is strictly localized in the parenchyma of the cornea. The tension and opacity of the rest of the cornea and the humors are not altered and admit of an ophthalmoscopic examination. There is no material interference with vision.

To clarify the etiopathogenic mechanism of the trouble, the authors sought to establish a relation between it and cholesteremia which, by means of Grigaut's colorimetric method, showed that cholesterol values of the blood did not differ materially from those of normal blood. However, the chemical examination of the lesioned tissues showed that the deposit consists of lipoidal substances, notably the esters of cholesterol, favored by the slowness of the interstitial circulation of the cornea. Invisible traces of distemper are perhaps the starting points.

The only successful intervention is ablation of the opaque spot. The use of vitamins (carotene, cod liver oil) is not effective. On the other hand, several subjects operated upon made good recoveries. Although rather delicate, the results are quite satisfactory. Under perfect local anesthesia the lesion can be scooped out by gentle strokes with a thin razor blade bent between the fingers. Mercurochrome 1:100 or cyanide of mercury are the preferable disinfectants and cocaine the best local anesthetic. A cholesterine solvent (ether or zylol) will aid the process of epithelization. (*V. Robin and A. Charton. La lipoidose corneenne du chien. Recueil de Médecine Vétérinaire, cxv, June 1939, pp. 321-331.*)

Poultry Mortality

The need of swinging the veterinary service into action in defense of infectious diseases of poultry is no longer a debatable question. Neither quackery nor science applied in hit or miss fashion will save the billion-dollar poultry industry from the hazards of disease. Of the 420,257,000 chickens on American farms in 1937, nearly 64,000,000 died during that year, exclusive of chick and young stock mortality.

The loss from adult mortality is set at $48,000,000, which does not include the

cost of maintenance up to the time of death. Moreover, the loss among adult hens is mounting while that among chicks has decreased through researches which have been put into practice. Hatchability and chick viability have likewise been augmented. On the other hand, laying-house mortality has increased with staggering rapidity. The reason lies in the inability of owners to detect disease before it has spread beyond control.

Except in the treatment of worm parasites and ectoparasites, drugs are of no great value to poultrymen, not at least to the same extent as in mammalian therapeutics. . . . The adult mortality of chickens is a challenge to the veterinarian, who is advised to somehow inform his clients that he has a service to offer in the handling of the farm flock. . . . A glance through the "poultry set-up" of a farm frequently gives a splendid opportunity to proffer the necessary preventive measures.

There is no longer any reason why coccidiosis, pullorum disease, tuberculosis, fowl pox, laryngotracheitis, cholera and typhoid should continue to take a heavy annual toll from the farm flocks. Adherence to the plans set up by the federal bureau of animal industry will enable the practitioner to aid in stopping these tremendous losses. *(J. Holmes Martin. Veterinary Medicine, xxxiv, December 1939, pp. 687-690.)*

Accurate Diagnosis of Poultry Diseases

If "reminiscing is a sure sign of senility," it is nevertheless important in clarifying the poultry situation to the present personnel of the veterinary profession. Poultry pathology is an infant science and poultry practice an art in the embryonic stage.

So little was known about poultry diseases only a few years ago that meager attention was paid to them in the veterinary colleges and, consequently, poultry practice was not thought to be a part of the veterinarian's obligation to his clients. Poultry was not considered sufficiently important to attract public attention to the need of appropriations for research and teaching. Poultry raising was a sideline and, therefore, the graduates went into the field uninformed and unimpressed.

A great change came about when the incubator and commercial hatchery supplanted the "little red hen" as the foster mother of chickdom. With this transformation came the great industry and with it the inevitable effect of agglomeration on the incidence of infectious diseases. A total loss of 70 per cent of chicks hatched is not unusual. "Ignorance always begets superstition," so there sprang up groups of self-styled poultry specialists without medical training who exploited "sure cures" in the absence of a technical veterinary service, notwithstanding that accurate diagnosis of the multiple infections affecting chickens is the preliminary necessity. The 17 organisms of the paratyphoid group, identified serologically, indicate the complexity of this problem alone, and the need of the laboratory for the isolation of the etiological agent.

The practitioner is urged to keep in touch with the passing events, since the value of poultry and poultry practice is great and "veterinary practice is to a large extent founded upon economic principles." *(C. P. Fitch and B. S. Pomeroy. Veterinary Medicine, xxxiv, December 1939, pp. 693-697.)*

Estrus in the Sow

Estrual cycles succeed one another regularly throughout the year. The duration of the cycle varies from 15 to 30 days (Craig). The average duration is three weeks. According to precise histological studies made recently by Karl Ammann in the course of his work on artificial insemination, the cycle is divided as follows:

Anestrus	8-10 days
Proestrus	3 days
Estrus	3 days
Metestrus	7 days

During heat, 6 to 15 follicles mature, and they rupture successively between the 30th and 48th hours of heat (Lewis). Milovanov recommended fecundation of the sow on the second day, and Quinlan, not before

the tenth hour. Stegu observed that in certain breeds, fecundation is more certain when sows are bred on the fourth day, that is, 24 hours after the end of estrus.

If a sow is not fecundated, the involution of the corpus luteum is quite slow; it lasts one or two months and is prolonged through several cycles. A large number of follicles suffer atresia in the course of every phase of the cycle, especially during metestrus.

After fecundation, the cycle is interrupted and estrus occurs two months after parturition, or, more precisely, one week after weaning (Craig, Stross, Struve). The exclusive presence of keratinized cells and the absence of polynuclears in the vaginal fluid are characteristics of heat in sows. (*Excerpt from Estrual Cycle in Mammifers [title translated]. Recueil de Médecine Vétérinaire, cxv, February 1939, p. 70.*)

BOOK NOTICES

Parasites of the Domestic Horse

The horse is notorious for its variety of parasites. In this respect it has no equal in the animal kingdom and, as a consequence, the parasitologist who sets out to cover the entire field must write a voluminous book. No English author has ever undertaken the task of grouping the protozoan and metazoan parasites injurious to horses. The literature on the subject is found scattered through general texts on parasites of animals. G. Carpentier, associate professor of parasitology of Alfort, added such a book to the literature when he wrote *Parasites et des Maladies des Equides Domestiques* (Parasites and Parasitic Diseases of the Domestic Horse), a volume of 524 closely written and well-illustrated pages of 6″ x 10″, without wandering beyond the scope indicated by the title.

In the foreword, Professor Henry, distinguished parasitologist and successor of Neumann, who wrote the first classic on the subject, compliments the author for continuing the life work of the latter, who envisaged the notion of classifying works on animal parasitology by groups. Thus, this work on the horse becomes a continuation of *Parasites et Maladies parasitaires des Oiseaux domestiques* (1909) (Parasites and Parasitic Diseases of Domestic Fowl) and *Parasites et Maladies parasitaires du Chien et du Chat* (1914) (Parasites and Parasitic Diseases of the Dog and the Cat).

Carpentier's book is distinguished for affectuating the practical side of the subject. Without dodging morphology and life histories and meticulous classification, it covers prophylaxis and therapeutics in minute details. In fact, "Equine Parasitisms and Their Treatment" would be an appropriate title, as nothing in the latter respect seems to have been omitted. Moreover, the book appears to consider all of the parasites affecting horses. (*Parasites et Maladies Parasitaires des Equides Domestiques. G. Carpentier, associate professor of parasitology, École Nationale Vétérinaire d'Alfort. 524 pages, profusely illustrated. Vigot Frères, Paris, 1939.*)

Stedman's Medical Dictionary

The first edition of this popular medical dictionary appeared in 1908, the 14th in 1939. Stedman's is well known in the veterinary circle for the author's determination to iron out the confusing orthography of medical terms through all of these years. While other medical lexicographers were swaying from one style of spelling to another (and back again), Stedman remained firm in his conviction that medical terms spelled in obedience to basic etymology could not for long be replaced by ill-conceived orthography. That considerable harm has been done to American medical literature by switching from one style to another without good reason is quite evident. Customs adopted in the editorial rooms of medical and veterinary journals widely circulated have left blemishes in medical English that are yet to be healed.

The arbitrary dropping of the *e* from the names of alkaloids, for example, still

lingers, notwithstanding that careful writers have long since ceased to commit that sin. An excellent way of distinguishing the alkaloids from the glucosides was lost when the *e* was dropped from such words as eserine and morphine. The same may be said of the shifting of *c* into *k* in words important to the veterinarian, such as leucemia and ancylostomiasis.

These examples demonstrate how faulty spelling leads to equally faulty pronunciation. Sibilant *c* became rough *k* in these and other words in everyday use. In short, on account of arbitrary notions of medical lexicographers, medical dictionaries have not consistently followed the dictionaries of the general language. From the standpoint of the purist, some medical dictionaries are a comedy of errors. Stedman's is an exception.

Inasmuch as veterinary medicine has trekked a long way since the turn of the century, terms peculiar to that field of medicine should either be omitted or else brought up to date. In view of the tremendous terminology that has crept into the language of veterinary medicine, it seems pardonable to advise the authors of medical dictionaries to court a closer acquaintance with animal pathologists. (*Stedman's Medical Dictionary, 14th edition, illustrated. By Thomas Lathrop Stedman, A.M., M.D., and Stanley Thomas Garber, B.S., M.D. The Williams and Wilkins Company, Baltimore, 1939.*)

Abundant U. S. A.

The United States, with a little over 15 per cent of the total land area of the world, produces 46 per cent of its cotton, 12 per cent of its wool, 27 per cent of its rayon, and 25 per cent of its oranges. Although we have only 6 per cent of the world's population, we own 70 per cent of its automobiles, 56 per cent of its telephones, and 35 per cent of the railroad mileage.—*The Commentator.*

Man lives longer than all mammals, including elephants and whales.—*Pathfinder.*

Love of Wild Life

I love the brooklets, I love the treelets, I love the flowerlets, as long as I don't have to weed them, and I am extremely fond of birds, particularly ducks done not over eleven minutes. . . . I have a keen interest in the mating habits and nesting haunts of the genus *homo.* In short, I am not above mingling with the human flock and beholding vanities, vulgarities, goodness, grafting, posing, piety and the rest of our wild life.—*Jesse Lynch Williams in Scribner's.*

Santonin for pinworms in the human being is less than 50 per cent effective when given by the mouth in daily doses for a period of ten days.—*Public Health Reports, November 10, 1939.*

The waterfowl population of the United States has doubled in the past five years; between 50 and 60 million birds follow the flyways.—*U. S. Bureau of Biological Survey.*

Out of 3 million surgical operations performed in the United States and Canada each year, less than 4 per cent result in death, in contrast with the last century, when 60 per cent of those operated on died. —*Science Service.*

Says the London correspondent to the *Journal of the American Medical Association,* "In London no one goes any distance without taking his gas mask, notwithstanding that Germany has agreed to observe the Geneva Protocol prohibiting the use of gas and bacteria in war."

A number of terms are used to describe the tails of various breeds of dogs. The tails of setters, for instance, are called flags; hound tails are sterns; the over-the-back appendages of Pomeranians and Pekingese are referred to as plumes; the Collie's tail often is called a brush; the short tail of the pug is a twist.—*Bob Becker in the Chicago Tribune.*

THE NEWS

Nation's Capitol Will Be Host to A.V.M.A., August 26-30, 1940

United States Capitol.

"VISIT THE NATION'S CAPITOL IN AUGUST"

At the December meeting of the Executive Board, the Mayflower Hotel in Washington, D. C., was selected as headquarters of the Association's 77th annual meeting and the dates were set officially as August 26-30, 1940.

Already, plans for extensive publicity are being made. Through news releases to the popular press and coast-to-coast radio talks by prominent veterinarians, the attention of people in every section of the country will be focused on the events of the convention, and the part of the veterinary profession in the national scheme will be forcefully told. The slogan, "Visit the Nation's Capitol in August," will be widely publicized within the profession to invite the attendance of all eligible veterinarians.

The local Committee on Arrangements, headed by John R. Mohler, will hold an important meeting in February, at which time a comprehensive schedule covering every detail of the convention will be planned. A. Eichhorn of Beltsville, Md., is associate chairman of the Committee and I. M. Cashell of Washington, D. C., secretary. The other members of the Committee are George W. Gillie, in charge of publicity; H. M. O'Rear, entertainment; Mrs. Clifton D. Lowe, ladies' entertainment; H. W. Schoening, educational exhibits; Joseph F. Crosby, technical exhibits; R. A. Kelser, banquet; A. E. Wight, alumni dinners; W. H. Wright, president's reception; John P. Turner, large animal clinic; Mason Weadon, small animal clinic; and Walter J. Hall, poultry and sheep clinic.

In the 77 years of the Association's existence this will be the second meeting held at the national capitol. The first was in 1891. R. S.

Huidekoper and W. Horace Hoskins were president and secretary, respectively. Both will be remembered as distinguished Pennsylvania educators. In contrast, it is interesting to note that the incumbents of these offices 50 years later (1940) won their status in the field of practice.

Associated Serum Producers Continuing Educational Campaign This Year

A 1940 educational campaign designed to teach livestock owners to make greater use of veterinary services was planned at a recent meeting in Kansas City, Mo., of member companies of the Associated Serum Producers. The organization thus continues the singularly successful program which it conducted during 1939 (and for several years previous) in the interests of the veterinary profession.

The present campaign, which is to be continuous through December of this year, will utilize more than 1,200 local newspapers, 116 radio stations, 7 farm magazines and other auxiliary avenues of education.

A.V.M.A. ACTIVITIES

With the coming of this issue, the election (by mail) of a member of the Executive Board for the seventh district will be started. The election is necessitated by the moving of W. A. Sullivan from Wyoming to Indiana. Since President Way decided not to appoint a successor to Dr. Sullivan, the winning candidate will take office at the beginning of the Washington meeting.

Ballots for the nominations will be mailed to all members of the district the first week of January and are returnable for counting within 60 days. The five nominees receiving the highest number of votes will be the candidates of the election. When the nominees have been thus chosen, a second ballot will be distributed, and the one among the five nominees receiving a plurality will be declared elected.

The seventh district comprises: Alaska, Idaho, Montana, Nebraska, North Dakota, Oregon, Philippine Islands, South Dakota, Washington and Wyoming. The number of members in these states varies from 1 in Alaska to 133 in Nebraska.

Voters who have not paid the dues for 1940 will please enclose $5.00 with their ballot.

• • •

Conforming to the plan of furnishing speakers to student chapters of the Association at the various colleges, Glenn L. Ebright of Hammond, Ind., addressed the Michigan State Chapter on clinical feline medicine in November.

• • •

Secretary Merillat addressed the Nebraska State Veterinary Medical Association, December 12-13, at Omaha on matters concerning the Association and Women's Auxiliary.

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Among the actions taken by the Executive Board at its semiannual session, held at the Morrison Hotel in Chicago, December 4, was the increase of the annual dues to $7.50. The change, provided it is approved by the House of Representatives at the Washington meeting, will go into effect January 1, 1941. Concurrent with this plan is the gradual enlargement of the JOURNAL to 192 pages, recommended by the Board of Governors.

• • •

The Executive Board voted to continue the Association's exhibit at the New York World's Fair during 1940. John R. Mohler, chairman of the veterinary exhibit committee, reported favorable financial prospects which will enable the committee to carry on for another year without material strain on the Association's budget. The financial report of the fair exhibit for last year and the estimated expenses and receipts for next year were read into the records of the Board meeting. This financial report will be published in the February issue.

• • •

A committee of the Executive Board consisting of O. V. Brumley, J. G. Hardenbergh and C. W. Bower met with a committee of the Institute of American Meat Packers on December 3 to discuss the question of issuing seals of approval for prepared dog foods in connection with the plan now in operation between the A.A.H.A. and the A.V.M.A.

• • •

President Way attended the New England Veterinary Medical Association meeting at Hartford, Conn., October 2-3. He spoke on "Lameness in the Horse" at the general session and on "A.V.M.A. Affairs" at the banquet. He spoke before the Eastern Iowa Veterinary Association at Cedar Rapids, October 17-18, and the Tri-State Veterinary Medical Association at Sioux City, Iowa, in November on "The A.V.M.A.—Your Association and Mine."

Dr. Way is scheduled to attend the California Veterinary Conference at Davis, January 3-6, and the Illinois State Veterinary Medical Association meeting at Springfield, February 15-16. He will be a guest speaker at the dedication ceremony of the new veterinary building at Colorado State College, Fort Collins, on February 20 and will attend the College's short course, to be held February 20-22.

• • •

President-Elect Wight joined Dr. Way at the New England meeting. He also attended the meeting of the Southern Veterinary Medical Association, November 9-11, at Columbia, S. Car. While in Columbia, Dr. Wight took part in a discussion of the A.V.M.A. with B. C. McLean of Aiken, S. Car., which was broadcast from the local station.

U. S. GOVERNMENT

Omaha Chapter of B.A.I. Assn. Proposes Publication of "Bureau Veterinarian" As Section of A.V.M.A. Journal

The Omaha chapter of the National Association of Bureau of Animal Industry Veterinarians passed the following resolution, November 27, 1939:

Inasmuch as there seems to be a feeling prevalent among the members of the Omaha chapter of the National Association of Bureau of Animal Industry Veterinarians that there should be a more united front among the veterinarians of America, we feel that much can be done to strengthen the relations between veterinarians of the bureau of animal industry and veterinarians of other fields. To bring this about we submit the following proposal:

1) That an endeavor be made to bring the membership of bureau veterinarians in the national association of bureau veterinarians and the American Veterinary Medical Association to 100 per cent.

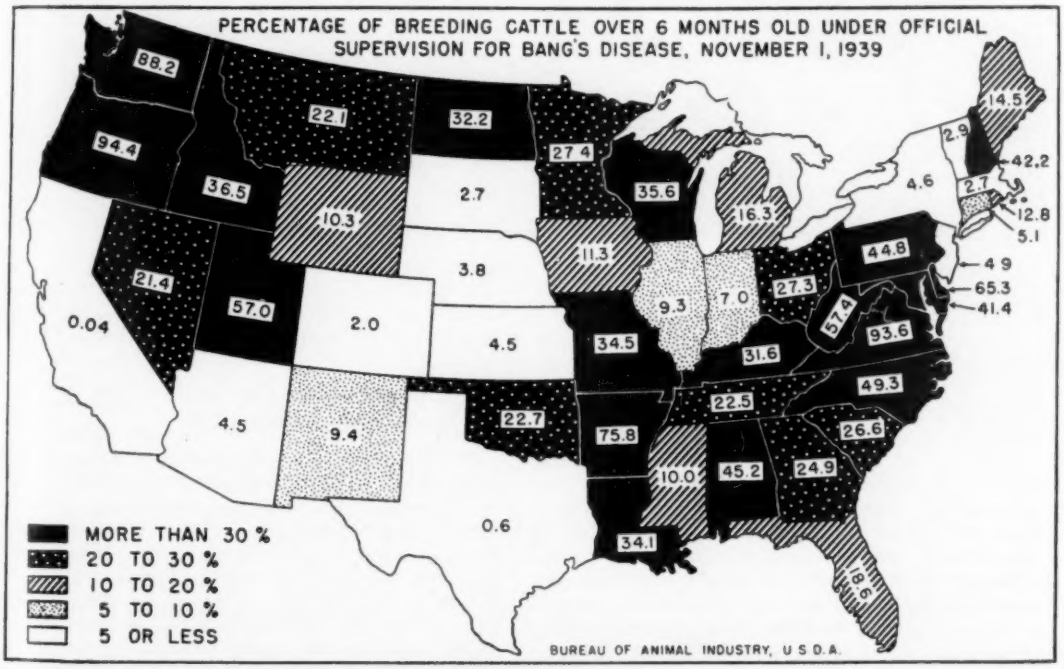
2) That the National Association of Bureau of Animal Industry Veterinarians edit the BUREAU VETERINARIAN to be published in a section of the *Journal of the American Veterinary Medical Association* set aside for this purpose.

We feel that an increased membership in the American Veterinary Medical Association would create a more friendly feeling between bureau and other veterinarians.

Also, if the bureau veterinarians are represented in the American Veterinary Medical Association in goodly numbers, when legislation favorable to the service is before Congress, the backing of the A.V.M.A. would be valuable.

Due to the vast increase in municipal meat inspection, we feel that articles edited in the BUREAU VETERINARIAN would be of great value and interest to the veterinary profession, espe-

More Herds of Cattle Under Bang's Disease Supervision



Cattle owners in the majority of states are showing continued interest in eradicating Bang's disease from their herds, officials of the federal-state campaign against this disease recently stated. The map above shows the percentage of breeding cattle over 6 months of age under supervision in each state on November 1, 1939. It will be observed that in seven states more than one half of the breeding cattle are under supervision in the campaign, which has been in progress since July 1934. The figures of this map compared with similar figures issued on May 1, 1939, show that many states have made substantial percentage gains. Greatest progress is recorded for Delaware, Georgia, Maryland, North Carolina, North Dakota, Oregon, South Carolina and Washington.

cially to the practitioner who must adjust himself to meet the demands of municipal meat inspection.

(Signed) C. J. PECHAL, *President.*

J. S. Jenison Feted by Chicago B.A.I. Force

J. S. Jenison (McK. '07), inspector in charge of meat inspection for the U. S. bureau of animal industry at Chicago, Ill., was the guest of honor at a banquet given by the bureau's Chicago employés at the Southmoor Hotel in Chicago, Saturday evening, November 18, 1939. The occasion was a farewell tribute to Dr. Jenison, who was transferred to bureau work at St. Louis, Mo., effective December 16, 1939.

Dr. Jenison's popularity in Chicago is attested by the fact that 250 members of the bureau staff in Chicago attended his farewell dinner, which, incidentally, was the largest gathering of bureau employés ever assembled. W. R. Kidwell was in charge of the arrangements and acted as toastmaster. Brief addresses were made by C. H. Elliott, representing the veterinarians; by W. A. Palmer, representing the lay inspectors; and by W. B. Hurd, representing the clerical force. Following the farewell address of Dr. Jenison, the remainder of the evening was devoted to music and entertainment provided by a number of talented members of the bureau staff.

The entire staff at Chicago is sincerely sorry to see Dr. Jenison leave their midst. During the 3½ years that he was in charge at Chicago, he won the respect and admiration of every one of the 300 employés under his supervision.

J. S. B.

More than 800,000 horses and mules were treated with chick-embryo vaccine during 1939, according to the federal bureau of animal industry. Of this number only 289, or 0.36 cases per 1,000 head, developed the disease. Among unvaccinated animals in the same areas, there were 1.3 cases per 1,000 head. More animals undoubtedly were vaccinated, since over 3,000,000 complete treatments were produced in 1939.

"Food and Life"

Food and Life is the title of the 1939 yearbook of agriculture, just off the press. In a single volume it deals with nutrition of human beings and live stock and comprises miscellaneous articles written by scientists of the U. S. Department of Agriculture.

This is the fourth of a series of yearbooks of agriculture dealing with farming. The 1936 and 1937 editions deal with genetics and breeding and are titled *Better Plants and Animals. Soils and Men* is the title of the 1938 edition.

Capt. Don L. Deane Is Winner Of Hoskins Medal for 1939

At the graduation exercises of the medical department professional service schools, held on November 30, 1939, at the Army Medical Center, Washington, D. C., Capt. Don. L. Deane of the veterinary corps, U. S. Army, was awarded the Hoskins medal. Sponsored by the A.V.M.A., this award is given to the student who attains the highest general average in the basic course at the Army Veterinary School.

Capt. Deane (Colo. '34) was commissioned a first lieutenant in the veterinary corps of the regular army on November 30, 1936, and promoted to the grade of captain on November 30, 1939. He served at Fort Bliss, Texas, prior to entering the Army Veterinary School in September. He is now enrolled in the Army Medical Field Service School at Carlisle Barracks, Pa., for a course of instruction at that post.

Regular Army

By direction of the president, and under the provisions of Public No. 18, 76th Congress, 1st Lieut. Howard Luther Kester, veterinary corps reserve, is ordered to active duty, effective November 20, 1939, and directed to proceed without delay from Cambridge, Neb., to Chicago Quartermaster Depot, Chicago, Ill., for duty.

Captain Robert A. Boyce, Jr., is relieved from assignment and duty at Fort Sam Houston, Texas, effective on or about November 1, 1939, and assigned to duty at Fort Riley, Kan.

Captain Velmer W. McGinnis is assigned to duty at Fort Benning, Ga., effective upon completion of his present tour of foreign service.

Captain William F. Collins is relieved from his present assignment and duty at the Army Veterinary School, Army Medical Center, Washington, D. C., effective upon completion of his present course of instruction, on or about November 30, 1939, and is then assigned to duty in the Panama Canal department. He will proceed to the New York Port of Embarkation, Brooklyn, N. Y., and report to the commanding general for temporary duty until such time as will enable him to sail on the transport leaving New York, N. Y., on or about March 5, 1940.

By direction of the president and under the provisions of Public No. 18, 76th Congress, Captain Robert Nixon Earhart, veterinary corps reserve, is ordered to active duty, effective December 5, 1939, and directed to proceed without delay from Columbus, Ohio, to Fort Des Moines, Iowa, for duty.

By direction of the president and under the provisions of Public No. 18, 76th Congress, 1st Lieut. William Sylva Gochenour, Jr., veterinary corps reserve, is ordered to active duty, effective December 7, 1939, and directed to proceed without delay from Indianapolis, Ind., to Camp Joseph T. Robinson, Ark., for duty.

Captain James B. Nichols will proceed from San Francisco, Calif., on temporary duty as transport veterinarian for the San Francisco-Panama portion of the trip of the USAT "Meigs," scheduled to sail from San Francisco on or about December 21, 1939. Upon arrival in Panama, Captain Nichols will return to his proper station.

Lieut. Colonel F. H. K. Reynolds is assigned to duty at Station Hospital, Fort Sam Houston, Texas, upon completion of his tour in Panama.

The following-named captains are directed to proceed from Washington, D. C., to Carlisle Barracks, Pa., and report to the commandant, Medical Field Service School, on or about December 2, 1939, for duty for approximately three months for the purpose of pursuing the basic course of instruction: S. G. Asbill, Walter Smit and Don L. Deane.

The promotion of the following-named officers to rank from date opposite their names is announced:

To Colonel.—Lieut. Colonel Christian W. Greenlee, November 26, 1939; Lieut. Colonel William H. Houston, November 27, 1939.

To Lieut. Colonel.—Major Jack K. Fuller, November 20, 1939.

To Major.—Captain Charles S. Greer, October 10, 1939; Captain John L. Owens, October 29, 1939.

To Captain.—1st Lieut. Don L. Deane, November 30, 1939.

Veterinary Corps Reserve

NEW ACCEPTANCES—FIRST LIEUTENANTS

Olin Alvin Anderson, 1027 Bellevue Court, Seattle, Wash.; Joseph Frederick Miller, Box 1487, Cristobal, Canal Zone; James Terrell Murphy, Cuthbert, Ga.; Clarence Andrew Woodhouse, P. O. Box 445, Angleton, Texas.

Leland D. Ives Elected President of N.Y.U.S.D.A. Club

Leland D. Ives (Amer. '96) of the agricultural marketing service, U. S. Department of Agriculture, was unanimously elected president of the New York section of the U. S. Department of Agriculture club on October 10, 1939.

Almost immediately after assuming office, Dr. Ives received a request from the board of education of New York City to speak before the students of the Food Trades Vocational High School about the Department's work. In fulfilment of the request, Dr. Ives presented a creditable summary of the Department's many activities and accomplishments in the New York area.

F.T.C. Rules on False Claims For Two Canned Dog Foods

In October of 1939, the Federal Trade Commission charged the Roxamma Canning Co. of Lebanon, Ohio, with falsely claiming that their Harty and Blackspot dog foods contain beef by-products.

In another action, the Commission caused the Joseph M. Julian Co. of Huntington Park, Calif., to cease claiming that their Marco dog food is "Best by Test," that it contains no starchy fillers, more meat or less starch than other competitive products, and that dog and cat food made in America contains more meat. —*Tide*, November 15, 1939.

A Salute to the Bureau of Animal Industry*

IT IS A PRIVILEGE for me to meet with you tonight. These annual gatherings of your organization and the United States Live Stock Sanitary Association, of which regulatory officials constitute the most important part of the membership, are noteworthy for their contributions to veterinary progress. I am happy to be able to claim among my closest friends many of the staff of the U. S. bureau of animal industry.

I know that most of you are longtime members of the American Veterinary Medical Association. Many of you have held high offices in the Association. Therefore, you will be as familiar as I with the fact that the A.V.M.A. has always aligned itself closely with the work of the B.A.I. I assure you that today, as ever, the veterinary profession of America is justly proud of you and the service you perform.

* * *

It is an undeniable fact that Secretary Wallace was absolutely correct when he made this statement recently, "The federal meat-inspection service has attained a plane of efficiency and protection unexcelled by that of any other nation."

* * *

The general theme of my brief remarks tonight is coöperation. There are matters in which the American Veterinary Medical Association can coöperate more closely with the official work in which you are engaged. Also, I have some suggestions about better coöperation which regulatory officials might render to the practitioner and his clients, the livestock breeders.

We are being severely criticized for arbitrary "hard-boiled" regulations regarding the interstate movement of animals from accredited and approved herds, and for lack of uniformity of results in the application and interpretation of scientific biological tests. Many breeders sincerely believe that we are withholding the results of field experiments and research that would be helpful to the average livestock owner in solving some of his problems. I refer to calfhood vaccination as a possible important aid in the control of Bang's disease.

Bang's disease and its many problems seem to be the chief bone of contention. The standard uniform antigen that is being prepared and distributed by the Bureau for coöperative testing will help to solve one important problem. If the 1:25 reading could be eliminated, it would do more to cause a better feeling and better relations with the livestock breeders than any single thing. At the present time a calf showing a 1:25 reaction in New York is considered healthy. Forty miles away, in New Jersey, the same animal at the same moment is considered diseased. This just does not make sense to the breeder of purebred cattle. It should not be necessary for a breeder who owns an approved and negative herd to send duplicate samples to three or four approved laboratories in order to get one negative test for interstate shipment of a 6-month-old bull calf. Accreditization means little, as blood samples have to be specially drawn for nearly every individual sale. Recently, 50 odd Guernseys were selected from accredited and approved herds to be sold at a breeders' consignment

*Presented at the annual banquet of the National Association of Bureau of Animal Industry Veterinarians, Morrison Hotel, Chicago, Ill., December 7, 1939, by Cassius Way, president of the A.V.M.A.

sale. When they were blood tested, 17 showed a 1:25 reaction and therefore were not eligible for sale. This is expensive and the fact is too often overlooked that most owners and breeders of healthy cattle make an honest endeavor to maintain clean herds, and they pay the bills. Unnecessary and excessive costs mean fewer breeders and less work for veterinarians.

Simplified, uniform regulations governing interstate shipment of livestock would be a welcome innovation. At the present time the various state laws and regulations make a volume consisting of 18 long, single-spaced typewritten pages. The length of time required to get interstate-shipping papers should be shortened. It takes far too long to ship into another state after making a sale. Many sales are lost due to this delay.

* * *

It is common knowledge that rabies and rabies control is one of the most controversial subjects now confronting the veterinary profession. Thousands of words have been written on this subject by dog writers, and heated discussions have been held wherever dog owners convene.

At the Memphis convention of the A.V.M.A. last August, a definite policy was unanimously adopted on this much debated problem. In the resolution the health officials of the nation were asked to turn over the control of canine rabies to the federal bureau of animal industry because it is a national problem. The Association declared that its policy would be on tenable ground only if it could present to the public the recommendation that the control of rabies b turned over to the veterinary profession and that the fundamental principles of the control of infectious animal diseases be applied to this disease. The fact that there is no rabies in England, in Australia or in most of the countries on the Continent was cited as an argument for the contention that rabies can be effectively controlled in this country.

The Association's resolution, in part, is as follows: While it is known that vaccination is not 100 per cent efficient, it is effective to a marked degree and can be used as an accessory agent of control. Fundamentally, however, we should hold to the principle that the disease can be controlled and will be controlled if it is turned over to the veterinary profession and the proper authorities qualified and prepared to execute an efficient control program.

I am happy to report that many of the writers of news about dogs in the nation's newspapers have endorsed the stand taken by the Association.

* * *

So much for the attitude of the press toward this proposal. To discover the attitude of the bureau of animal industry I have discussed the matter with Dr. Mohler. He tells me that the bureau would be quite willing to take over the control of rabies—even though it may be one of the most difficult problems yet faced—if the health officials of the nation approve the step.

AMONG THE STATES

Alabama

An interesting variation of the additional seven days of field training ordered for the National Guard was recently carried out by troop C (veterinary) of the 123rd medical squadron, 23rd cavalry division, under command of Capt. A. L. Faulk, V. C., of the Alabama National Guard.

On December 3, the unit, now motorized, mobilized at its armory in Samson, Ala., proceeded by truck to Auburn, and established itself in the cantonment recently vacated by a CCC soil erosion company.

The troop, completely organized as to messing and other facilities, spent the forenoons in camp routine and other military training and the afternoons, through the coöperation of Dean I. S. McAdory and the faculty of the School of Veterinary Medicine, Alabama Polytechnic Institute, in observing the clinics, dissecting rooms and other technical laboratories of the institution. Particular attention was paid to practice in equine restraint and to first aid treatment of various wounds. On December 9, the organization broke camp and returned to its armory.

The troop, consisting of two officers and 34 enlisted men, is one of the few completely organized units of its type. First Lieut. R. A. Johnson, V. C., of Opp, Ala., is second in command and the enlisted personnel is headed by First Sergeant A. L. Faulk, Jr., the son of the troop commander. The officers are both prominent veterinarians of southern Alabama, alumni of Auburn and members of the Alabama and the American Veterinary Medical Associations. The enlisted personnel is made up of young men largely of Geneva county, Ala.

Arizona

The Arizona Veterinary Medical Association held its annual meeting at the Adams Hotel in Phoenix, December 9. The object of the session was to reconstruct the activities of the Association, which had not been receiving the support of eligible veterinarians. Much interest was manifested at this meeting, however, and plans were made for expanding the scope and influence of the group. Seventy per cent of the veterinarians eligible for membership were present. Many traveled from remote parts of the state for the occasion.

Officers were elected for the ensuing year as follows: F. D. McMahon of Phoenix, presi-

dent; G. G. Crosbie of Tucson, vice-president; and D. Miller of Phoenix, secretary-treasurer.

J. B. McQUOWN,
A.V.M.A. Secretary for Arizona.

California

The 15th annual report of the Los Angeles county livestock department is a comprehensive document that every veterinarian could read to advantage. Leslie M. Hurt, prominent California member of the profession, is in charge of the department.

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A species of mosquito capable of transmitting equine encephalomyelitis to humans has been found in California, it was reported at the tenth annual conference of mosquito abatement officials, December 12, at Berkeley.

W. B. Herms, head of the University of California entomology department, said that the species (= *Aedes nigromaculis*) is multiplying rapidly in the state.

Colorado

Four Colorado veterinarians, all practitioners, each have a son studying veterinary medicine at Colorado State College, Fort Collins. They are G. G. Miller (Chi. '11) of Denver, M. E. Spratlin (Colo. '18) of Littleton, Jay H. Bouton (Colo. '25) of Aurora, and S. W. Beggs (Colo. '15) of Lamar.

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The Colorado Veterinary Medical Association has undertaken a project involving the compilation of fees charged by members of the profession within the state and the establishment, if possible, of a schedule of fees which will be acceptable to all.

Delaware

The annual meeting of the Eastern Guernsey Breeders Association was held at the Du Pont Hotel in Wilmington, December 12. M. A. Emmerson of the School of Veterinary Medicine, University of Pennsylvania, spoke on x-ray therapy in actinomycosis and C. C. Palmer of the University of Delaware, Newark, presented an illustrated lecture on mastitis.

District of Columbia

The Eighth American Scientific Congress will be held in Washington, May 10-18, 1940, under the auspices of the federal government.

Pursuant to a special act of the U. S. Congress, invitations on behalf of the president have been extended to the governments of the American-Republics members of the Pan American Union to participate in the meeting. Scientific institutions and organizations are also invited to send representatives.

Georgia

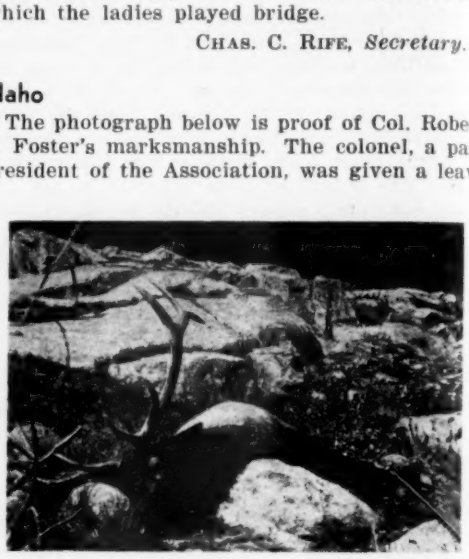
On November 3, H. C. Smith of Fort Dodge, Iowa, gave an illustrated talk before the Atlanta Veterinary Medical Society on fungus diseases in dogs and cats. Gastroenteritis in cats was discussed at length by Dr. Smith and all members of the group.

Preceding the lecture, the veterinarians and their wives enjoyed a fine dinner, following which the ladies played bridge.

CHAS. C. RIFE, *Secretary.*

Idaho

The photograph below is proof of Col. Robert J. Foster's marksmanship. The colonel, a past president of the Association, was given a leave



The "beeg" bull elk

of absence from the Presidio of San Francisco, Calif., early in October to spend a few days at Idaho's Indian Park hunting grounds. The "beeg" bull elk (as Col. Foster described him) pictured here was shot close by Martin creek. The antler spread is 47⅝ in. Indian Park is located about 100 miles east and south of Lewiston.

Illinois

Four applicants, all from rural districts, were licensed to practice at the state board examinations held in Chicago, December 18-19. This represents a change from recent years, when the large percentage of veterinarians licensed in the state were destined to small animal practice in the large centers of population. The change seems to indicate that Illinois has again become a desirable place to enter the practice of veterinary medicine. Those licensed were C. R. Collins (Iowa '28) of Dixon; C. A. Poppenhouse (K. S. C. '39) of Ashton; D. C. Vest (Iowa '37) of Chapin; and W. J. McAlister (K. S. C. '38) of Wheaton.

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At the annual meeting of the Horse and Mule Association of America, held at the Palmer House in Chicago, December 6, Louis E. Stoddard of New York City, internationally famed polo player, was elected president of the organization; Grant Good, Belgian breeder of Ogden, Utah, was chosen first vice-president;

W. H. Weeks, general manager of the Kansas City stock yards, second vice-president; and F. M. Holmes of New Britain, Conn., treasurer. Wayne Dinsmore continues as executive secretary.

• • •

Forty-one veterinarians attended a meeting of the Central Illinois Veterinary Association on November 9 at the Leland Hotel in Springfield. A. E. Dickerson of Springfield was elected president.

A resolution was adopted to the effect that the state veterinary department be transferred from the department of agriculture to the department of health. A corollary resolution adopted at the meeting provides for the rewriting and modernizing of the state practice act, the revised document to be presented for the approval of the executive board of the state association.

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Among the host of distinguished speakers who participated in the program of the 43rd annual meeting of the United States Live Stock Sanitary Association was Etienne Létard, professor of zoötechnics in the National Veterinary School at Alfort, France.

In his address before the Association, which convened in Chicago at the Morrison Hotel, December 6-8, Professor Létard lauded Amer-

John R. Mohler (left) and Professor Etienne Létard.

ican veterinarians for their accomplishments in animal-disease eradication and control.

H. P. Rusk, longtime friend of the veterinary profession and dean of the College of Agriculture, University of Illinois, Urbana, delivered the address of welcome which opened the three-day proceedings.

Iowa

Thirty-seven veterinarians attended a dinner meeting of the Cedar Valley Veterinary Association at Black's tea room in Waterloo, December 11.

One of the features of the meeting was a question box on practice problems. This stimulated a lively discussion, particularly on obstetrics, swine-disease control and mastitis.

C. E. STRAIN, *Secretary.*

Kentucky

The International Salmonella Center, Copenhagen, Denmark, has designated the agricultural experiment station of the University of Kentucky, Lexington, as the American Salmonella Center for the identification of diseases, it was announced recently.

The Kentucky station was selected for this project because of its extensive work in disease control, including the studies and investigations conducted by P. R. Edwards of the department of animal pathology. Dr. Edwards has typed approximately 500 bacteria cultures, and the department has been supplied with 100 cultures from the International Salmonella Center.

Massachusetts

The October meeting of the Massachusetts Veterinary Association was one of the largest held by the Association for some time. The speaker of the evening, Newton C. Browder, a surgeon of the Boston City Hospital, showed excellent color films of a number of operations and his accompanying lecture brought out a number of technics applicable to veterinary practice.

Michigan

The semiannual meeting of the Michigan-Ohio Veterinary Medical Association was held at the Masonic Temple in Blissfield, November 15. T. J. Hage of Monroe presided.

Preceding the meeting, a clinic was held at the hospital of J. C. Schwabland, where E. S. Weisner, W. H. Beck and A. M. Jacoby conducted the rapid-method agglutination blood test and a tuberculin test of a flock of hens. This demonstration was designed to show how the rapid test can be conducted by veterinarians in farm poultry houses. The tables used in the demonstration were designed and built by Dr. Jacoby.

Contributors to the literary program were A. F. Calkins of Lansing; E. S. Weisner of Michigan State College, East Lansing; B. J. Killham of Michigan State College; and L. D. Barnhart of Michigan State College.

Minnesota

The fall quarterly meeting of the South Central Minnesota Veterinary Medical Association was held at Faribault on October 18, Ralph West of Waseca presiding.

W. L. Boyd of the University of Minnesota, St. Paul, presented a paper on breeding diseases of cattle. This was followed by a round-table discussion of problems in cattle practice.

Montana

W. J. Butler of Helena, state veterinary surgeon, was elected president of the Montana Public Health Association at the organization's annual meeting.

Nebraska

A unique departure in house-organ publication is the special holiday issue of *The Norden News*, totally devoid of advertisements. Folklore about Christmas for grown-ups and children, two popular odes, a message of holiday greetings illustrated by pictures of the Norden staff, and culinary suggestions for the Christmas dinner make up the covers and outer pages. But, the surprise is the body of the issue, portraying a pictorial history of the existing veterinary schools of North America—one page for each, including an excellent picture of the main building, a recent portrait of the dean, and a brief but quite complete story of the institution. As the newspapers say, "It's a scoop."

New York

The Long Island Veterinary Medical Association held its December meeting at Huntington, L. I., on December 14.

Gerry B. Schnelle of the Angell Memorial Animal Hospital, Boston, Mass., spoke on radiography. He demonstrated numerous plates that he had taken of various conditions at different times and explained how they aided in the diagnosis of prevailing ailments.

An election of officers was held, with the following results: President, Kenneth F. Hilbert of Amityville, L. I.; vice-president, Arthur W. Frederick of Northport, L. I.; and secretary-treasurer, Herman Tax (reëlected) of Farmingdale, L. I.

Dinner was served after the meeting.

HERMAN TAX, *Secretary.*

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A regular monthly meeting of the Veterinary Medical Association of New York City was held at the Hotel New Yorker in New York City, November 1, J. B. Engle of Summit, N. J., presiding.

Col. R. A. Kelser of the veterinary corps, U. S. Army, addressed the group on the subject of rabies. A lively discussion followed Col. Kelser's talk, Drs. Danziger, Finkelstein and Barto participating.

Case reports were presented by E. S. Herman, G. F. Kinsey and J. F. De Kiralyi.

J. J. MERENDA, *Secretary.*

Ontario

The Ontario Veterinary Association has become a component association of the A.V.M.A. through the action of its executive committee in November. This is the second province of the Dominion of Canada to affiliate. The other is British Columbia, announced in the November 1939 issue. W. J. Rumney of Hamilton, Ont., is the secretary and registrar. Up to the present time Canada has been represented in the councils of the Association by a member of the Executive Board only. The Dominion associations in the several provinces did not avail themselves of the opportunity to be represented in the House of Representatives until this year.

Pennsylvania

R. Adams Dutcher, head of the department of agricultural and biological chemistry at Pennsylvania State College, has been appointed to fill a vacancy on the scientific advisory council of the committee on foods of the American Animal Hospital Association, in whose program of food testing the A.V.M.A. is coöperating.

Rhode Island

During the 1939 legislative session the Rhode Island Veterinary Medical Association succeeded in changing their veterinary practice act. A new act was passed by the assembly. During the 1940 session of the assembly, the Association hopes to introduce an amendment of the present act.

Texas

In a recent edition of the *Texas Veterinary Bulletin*, which is edited by M. B. Starnes of Dallas, it was stated that the Texas state association is waging a determined fight to control the veterinary activities of unlicensed offenders. This effort has at the same time brought out the fact that a little "housecleaning" is necessary within the ranks of the licensed veterinarians in that state.

This move on the part of the state association shows convincingly that the Texas organization is interested in the welfare of the profession—that it is not on any count a "back-patting society."

COMING MEETINGS

Small Animal Hospital Association. Los Angeles, Calif. January 2, 1940. R. W. Gerry, secretary, 8474 Melrose Ave., Los Angeles, Calif.

New York City, Veterinary Medical Association of. Hotel New Yorker, New York, N. Y. January 3, 1940. J. J. Merenda, secretary, 136 W. 53rd St., New York, N. Y.

Ohio State Veterinary Medical Association. Deshler-Wallick Hotel, Columbus, Ohio. January 3-5, 1940. R. E. Rebrassier, secretary, Ohio State University, Columbus, Ohio.

California Veterinary Conference (Division of Veterinary Science, University of California, and California State Veterinary Medical Association coöperating). College of Agricul-

ture, University of California, Davis, Calif. January 3-6, 1940. Charles J. Parshall, secretary, 319 B St., Petaluma, Calif.

Dallas-Fort Worth Veterinary Medical Society. Fort Worth, Texas. January 4, 1940. H. V. Cardona, secretary, 2736 Purington Ave., Fort Worth, Texas.

Houston Veterinary Association. Houston, Texas. January 4, 1940. W. T. Hufnall, secretary, 1612-14 E. Alabama Ave., Houston, Texas.

Ak-Sar-Ben Veterinary Medical Association. Fontenelle Hotel, Omaha, Neb. January 8, 1940. J. D. Ray, secretary, 1124 Harney St., Omaha, Neb.

Minnesota State Veterinary Medical Society. Hotel Radisson, Minneapolis, Minn. January 8-9, 1940. H. C. H. Kernkamp, secretary, University of Minnesota, St. Paul, Minn.

Intermountain Livestock Sanitary Association. Salt Lake City, Utah. January 8-10, 1940. D. E. Madsen, secretary, Utah Experiment Station, Logan, Utah.

Chicago Veterinary Medical Association. Hotel Sherman, Chicago, Ill. January 9, 1940. W. A. Young, secretary, 157 W. Grand Ave., Chicago, Ill.

Indiana Veterinary Medical Association. Severin Hotel, Indianapolis, Ind. January 9-11, 1940. Charles C. Dobson, secretary, New Augusta, Ind.

Southeastern Michigan Veterinary Medical Association. Medical Arts Bldg., 3919 John R St., Detroit, Mich. January 10, 1940. F. D. Egan, secretary, 17422 Woodward Ave., Detroit, Mich.

St. Louis District Veterinary Medical Association. Melbourne Hotel, St. Louis, Mo. January 10, 1940. J. P. Torrey, secretary, 610 Veronica Ave., East St. Louis, Ill.

Willamette Valley Veterinary Medical Association. Albany, Ore. January 10, 1940. T. Robert Phelps, secretary, 1514 Washington St., Oregon City, Ore.

Wisconsin Veterinary Medical Association. Madison, Wis. January 10-11, 1940. B. A. Beach, secretary, University of Wisconsin, Madison, Wis.

Cornell University Annual Conference for Veterinarians. New York State Veterinary College, Ithaca, N. Y. January 10-12, 1940. William A. Hagan, dean, New York State Veterinary College, Cornell University, Ithaca, N. Y.

Texas, State Veterinary Medical Association of. San Antonio, Texas. January 11-12, 1940. M. B. Starnes, secretary, City Hall Annex, Dallas, Texas.

Maine Veterinary Medical Association. Orono, Me. January 12, 1940. A. E. Coombs, secretary, 1 Kennebec St., Skowhegan, Me.

Vermont Veterinary Medical Association. Hotel Vermont, Burlington, Vt. January 13, 1940. G. N. Welch, secretary, 43 Union St., Northfield, Vt.

Kansas City Veterinary Medical Association. Kansas City, Mo. January 15, 1940. S. J. Schilling, secretary, Box 167, Kansas City, Mo.

San Diego County Veterinary Medical Association. Zoölogical Research Bldg., Balboa Park, San Diego, Calif. January 15, 1940. Paul D. DeLay, secretary, State Poultry Pathological Laboratory, Balboa Park, San Diego, Calif.

Mississippi State Veterinary Medical Association. Alacza Hotel, Clarksdale, Miss. January 15-16, 1940. E. H. Durr, secretary, Clinton Blvd., Jackson, Miss.

District of Columbia Veterinary Medical Association. Mayflower Hotel, Washington, D. C. January 16, 1940. Wm. M. Mohler, secretary, 5508 Nebraska Ave. N. W., Washington, D. C.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. January 17, 1940. Charles Eastman, secretary, 725 S. Vancouver Ave., Los Angeles, Calif.

New Jersey, Veterinary Medical Association of. Hotel Douglas, Newark, N. J. January 16-17, 1940. J. G. Hardenbergh, secretary, c/o Walker-Gordon Laboratory Co., Plainsboro, N. J.

Kansas Veterinary Medical Association. Jayhawk Hotel, Topeka, Kan. January 17-18, 1940. Chas. W. Bower, secretary, 1128 Kansas Ave., Topeka, Kan.

Long Island Veterinary Medical Association. Long Island, N. Y. January 18, 1940. Herman Tax, secretary, State Institute, Farmingdale, Long Island, N. Y.

North Carolina Short Course for Graduate Veterinarians. North Carolina State College, Raleigh, N. Car. January 22-25, 1940. A. A. Husman, 320 Agricultural Bldg., Raleigh, N. Car.

Post-Graduate Short Course for Veterinarians. Veterinary Hospital, Michigan State College, East Lansing, Mich. January 22-26, 1940. Ward Giltner, dean, Division of Veterinary Science, Michigan State College, East Lansing, Mich.

Iowa Veterinary Medical Association. Hotel Des Moines, Des Moines, Iowa. January 23-25, 1940. C. J. Scott, secretary, Knoxville, Iowa.

Keystone Veterinary Medical Association. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa. January 24, 1940. C. S. Rockwell, secretary, 4927 Osage Ave., Philadelphia, Pa.

Massachusetts Veterinary Association. Hotel Westminster, Copley Sq., Boston, Mass. January 24, 1940. H. W. Jakeman, secretary, 44 Bromfield St., Boston, Mass.

Ontario Veterinary Association. Royal York Hotel, Toronto, Ont. January 25-26, 1940. W. J. Rumney, secretary, 612 King St. W., Hamilton, Ont.

Missouri Veterinary Medical Association. Columbia, Mo. January 31-February 2, 1940. J. L. Wells, secretary, 1817 Holmes St., Kansas City, Mo.

Connecticut Veterinary Medical Association. Hartford, Conn. February 1, 1940. Geo. E. Corwin, secretary, State Office Bldg., Hartford, Conn.

Virginia State Veterinary Medical Association. Hotel John Marshall, Richmond, Va. February 8-9, 1940. A. J. Sipos, secretary, 1102 State Office Bldg., Richmond, Va.

Illinois State Veterinary Medical Association. Leland Hotel, Springfield, Ill. February 15-16, 1940. C. C. Hastings, secretary, Williamsville, Ill.

State Board Examination

Oklahoma Board of Veterinary Medical Examiners. State Capitol Bldg., Oklahoma City, Okla. January 10-11, 1940. W. C. McConnell, secretary, Holdenville, Okla.

PERSONAL NOTES

R. P. Wagers (O. S. U. '36) has been appointed assistant professor of veterinary pathology at Kansas State College, Manhattan, Kan.,, succeeding C. C. Morrill (Mich. '33).

Merwyn P. Chapman (K. S. C. '38) has resigned from the service of the federal bureau of animal industry and is now engaged as pathologist and bacteriologist with the Idaho state bureau of animal industry at Boise.

L. E. Stanton (Iowa '25) of Jackson, Minn., was recently appointed a member of the board of the Southwestern Minnesota Sanatorium at Worthington, Minn. The institution treats human tuberculosis.

T. A. Ladson (U. S. C. V. S. '05) of Olney, Md., has taken into his practice his sons, Jack A. and T. A., Jr., both graduates of the University of Pennsylvania, class of '39.

R. C. Sweetser (O. S. U. '39) recently took over the practice of the late C. E. Dornheim at Mt. Airy, Md.

M. O. Robinson (U. P. '25), professor of bacteriology at the Alabama Polytechnic Institute, Auburn, Ala., was made a fellow of the American Public Health Association at the organization's 68th annual meeting in Pittsburgh, Pa., October 15-20, 1939. A.P.H.A. fellowships are conferred upon persons who have done meritorious work in the field of public health.

DEATHS

Hugh Frederick Dailey, for 17 years chief veterinarian at the Angell Memorial Animal Hospital, Boston, Mass., died of a heart attack in the Massachusetts General Hospital on October 29, 1939.

Born in New Haven, Conn., August 1, 1892, Dr. Dailey received his veterinary education at the University of Pennsylvania, from which he was graduated in 1913. He joined the staff of the Angell Memorial Animal Hospital when it opened in February of 1915 and became chief of staff in June 1922. A pioneer in small animal medicine, Dr. Dailey was one of the first American veterinarians to undertake the study and treatment of feline diseases.

Hugh F. Dailey

Dr. Dailey served overseas during the World War as a first lieutenant in the 183rd infantry. At the time of his death he was a member of the A.V.M.A. (joined 1926), the New England Veterinary Association, the Massachusetts Veterinary Association, and the Society of Phi Zeta. G. B. S.

Chauncey McCandless of Salem, Ohio, died on October 14, 1939, after an illness of several years.

Born at Prospect, Pa., on November 11, 1892, Dr. McCandless was graduated from the Chicago Veterinary College in 1915. Following service in the World War, he practiced in Lisbon, Ohio, until 1928, when he moved to Salem. He was at one time state veterinarian of Ohio.

Dr. McCandless joined the A.V.M.A. in 1918.

Wilfred B. Massie of Boston, Ind., was found dead in the basement of his home, of a shotgun wound in the chest, November 11, 1939. A shotgun was found near the body by the housekeeper when she went to fire the furnace.

Born on March 13, 1893, at Bessemer, Mich., Dr. Massie was graduated from the Michigan State College in 1916. He joined the Association in 1919.

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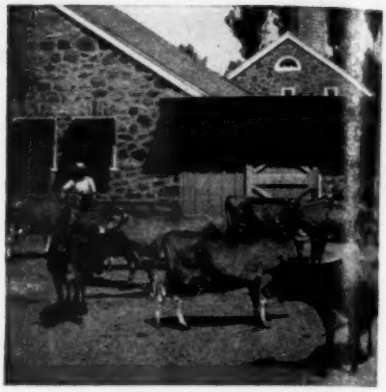

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